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DECEMBER 1960
VOL. 2 NO. 12

Journal of Occupational Medicine

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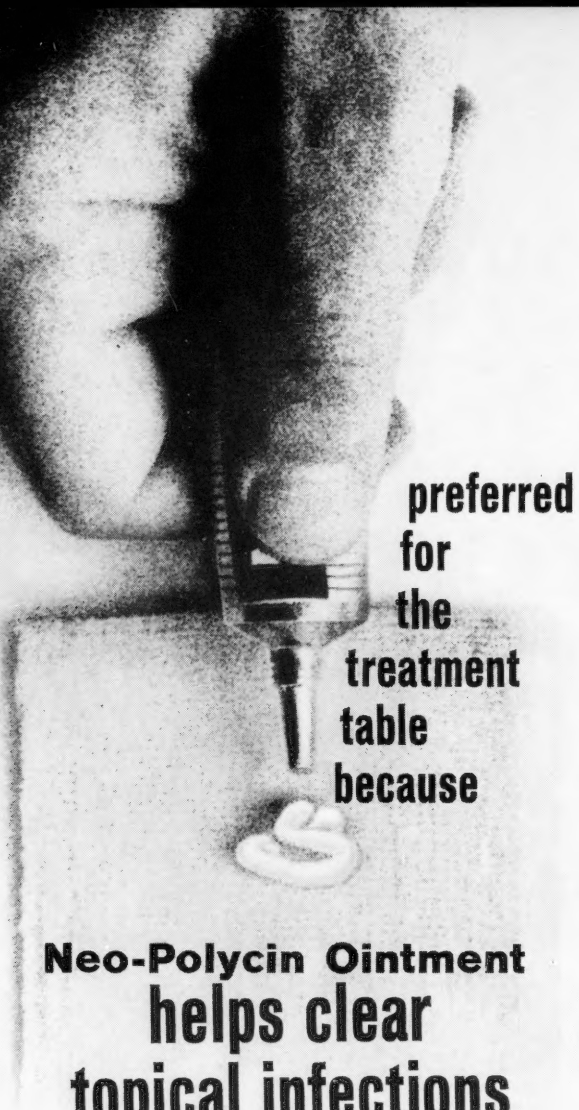
OFFICIAL PUBLICATION OF THE INDUSTRIAL MEDICAL ASSOCIATION



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Vol. 2 No. 12

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Official Publication of the Industrial Medical Association

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The Industrial Medical Association was organized in 1915 as the American Association of Industrial Physicians and Surgeons. Its first annual meeting was held the following year. In 1951 the present name was adopted.

The purposes of the organization as stated in the certificate of incorporation (not-for-profit) are:

"The object of this Association will be to foster the study and discussion of the problems peculiar to industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes of the medical care for employees; and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for their mutual advancement in the practice of their profession."

Presently, the membership includes more than 4000 physicians in the United States, Canada, Latin America, and other parts of the world. Physicians "who have an interest in Industrial Medicine" may apply for membership; specialization in this field is not essential. Regionally, the Industrial Medicine Association has 17 Component Societies in the U.S. The majority of the Association's members carry their memberships through one of the component groups.

The *Journal of Occupational Medicine*, owned and published by the Industrial Medical Association, is sent monthly to each member as one of the advantages of membership.

The annual business meeting and convention is held in the spring. Since 1945, the convention has been held concurrently with the meetings of other organizations having related interests including the American Association of Industrial Nurses, Inc., and the American Association of Industrial Dentists. This combination of meetings is known as the American Industrial Health Conference.

The Occupational Health Institute, Inc., is a non-profit educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the Occupational Health Institute has conducted the program of evaluation, approval and certification of Medical Services in Industry, on the basis of standards established by the Standards Committee of the Industrial Medical Association. This program was conducted by the American College of Surgeons for many years until its transfer to the Association.

The Association's headquarters office serves as a focal point for the Association's activities and through its coordination of information regarding industrial health problems encountered throughout the country, it offers invaluable assistance to members on a host of specific subjects.

ANNUAL MEETING—INDUSTRIAL MEDICAL ASSOCIATION and AMERICAN INDUSTRIAL HEALTH CONFERENCE

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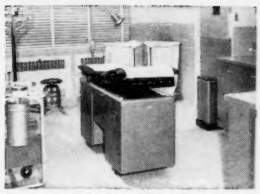

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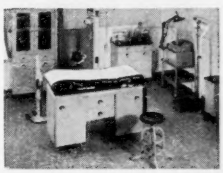

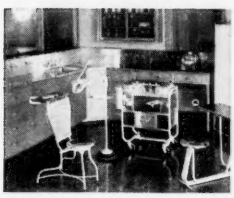

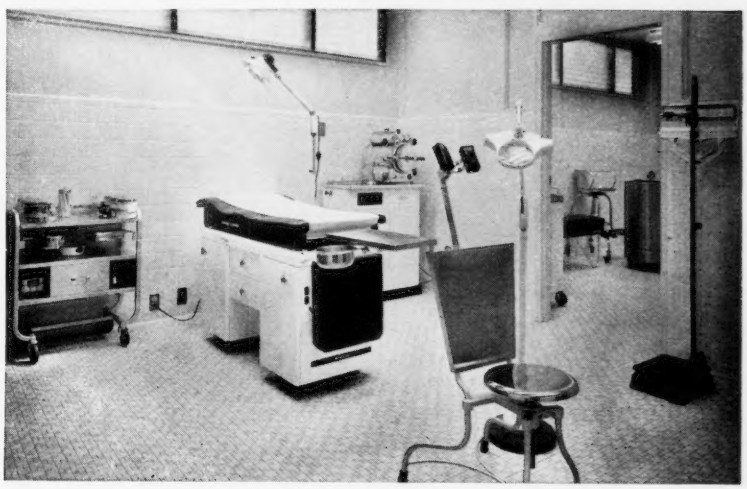

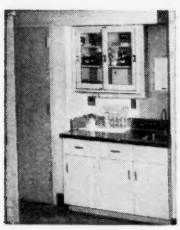

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1. Greene, C. C., Jr., and Beaver, G. T.: Journal of Occupational Medicine *2:363, 1960. 2. Influenza Fact Sheet: Public Health Service, U. S. Dept. of Health, Education and Welfare, August 1, 1960. 3. Burney, L. E.: Correspondence,* J.A.M.A. *174:438, 1960.*

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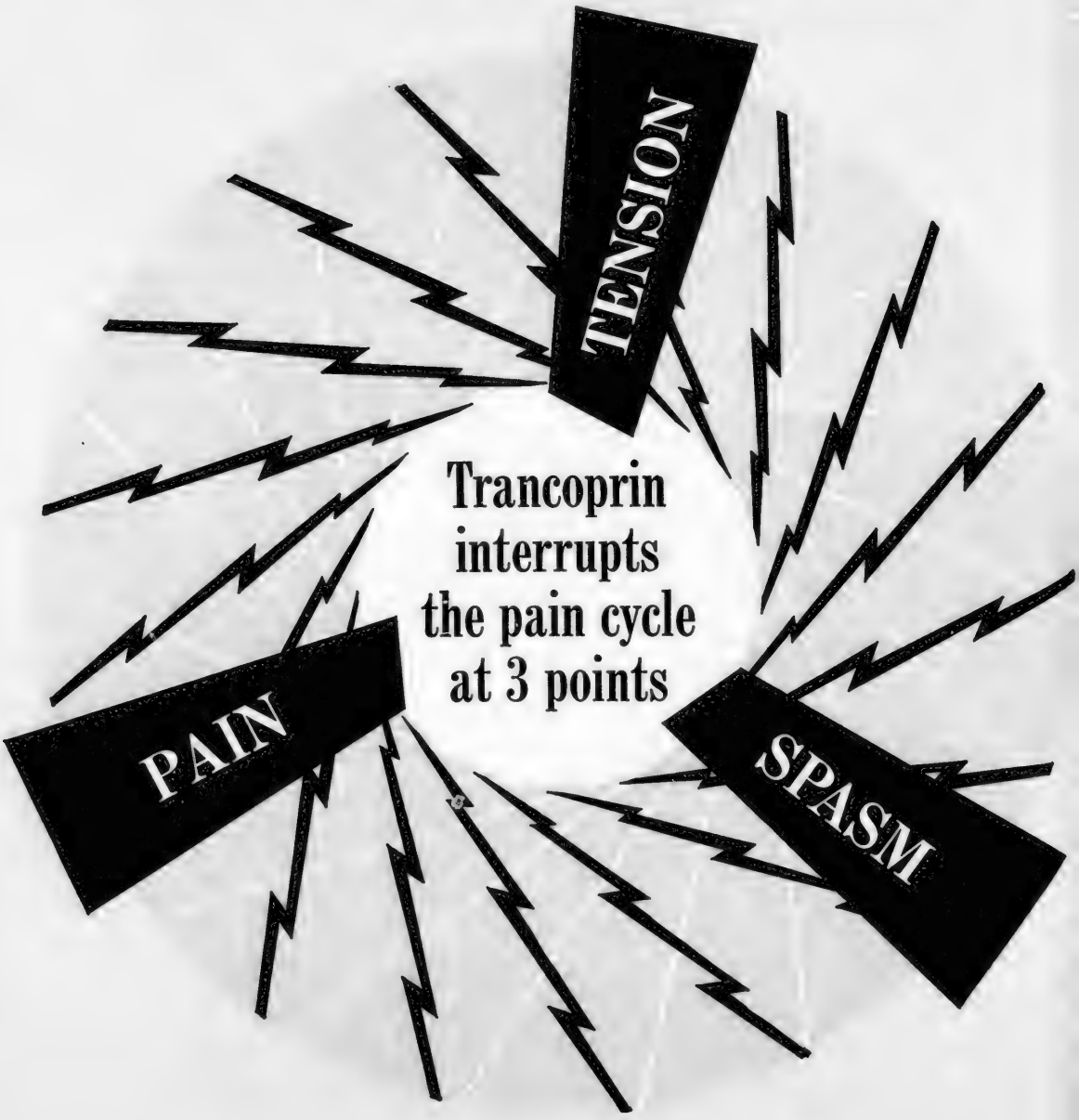

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References: 1. DeNyse, D. L.: *M. Times* 87:1512, Nov., 1959. 2. Ganz, S. E.: *J. Indiana M. A.* 52:1134, July, 1959. 3. Gruenberg, Friedrich: *Current Therap. Res.* 2:1, Jan., 1960. 4. Kearney, R. D.: *Current Therap. Res.* 2:127, April, 1960. 5. Lichtman, A. L.: *Kentucky Acad. Gen. Pract. J.* 4:28, Oct., 1958. 6. Mullin, W. G., and Epifano, Leonard: *Am. Pract. & Digest Treat.* 10:1743, Oct., 1959. 7. Shanaphy, J. F.: *Current Therap. Res.* 1:59, Oct., 1959. 8. Collective Study, Department of Medical Research, Winthrop Laboratories. 9. Hergesheimer, L. H.: An evaluation of a muscle relaxant (Trancopal) alone and with aspirin (Trancoprin) in an industrial medical practice, to be submitted.

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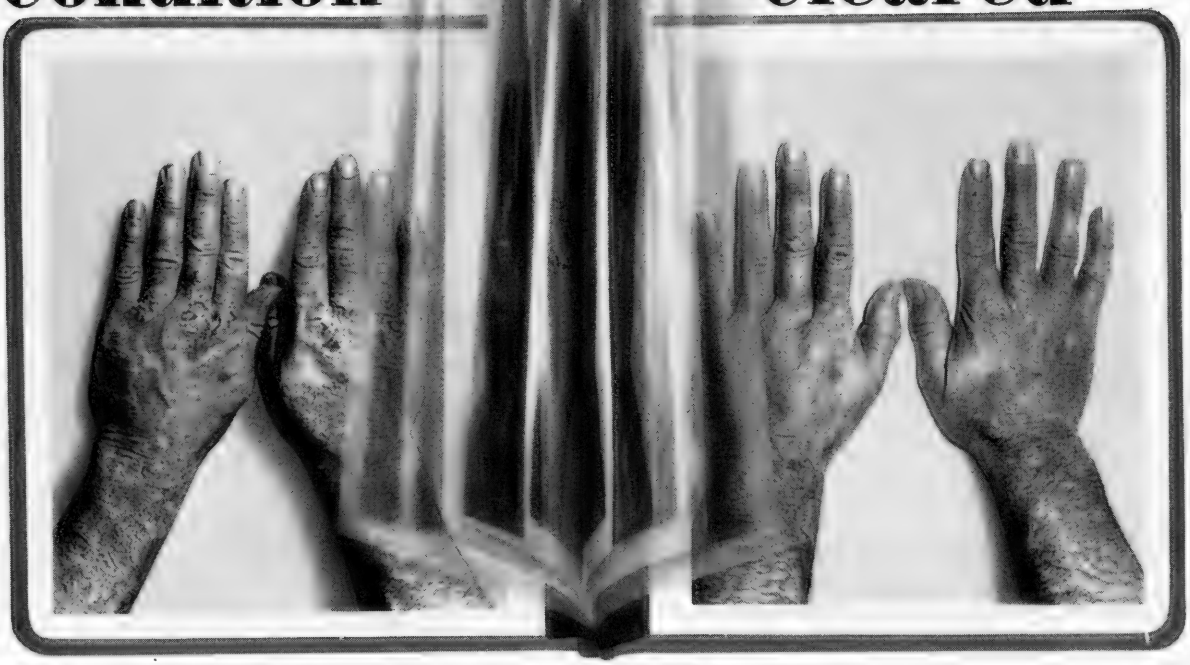

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*Dr. R. J. Murray, Medical Dept., Sperry Gyroscope Co., in an article published in the New York State Journal of Medicine, August 15, 1953 (Vol. 53, No. 16).

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Recommended Daily Dietary Allowances and the Nutritional Contribution of a Basic Cereal and Milk Moderate Low-Fat Breakfast*

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Whole Milk—4 oz.; Sugar—1 teaspoon;
Toast (white, enriched)—2 slices;
Butter—5 gm. (about 1 teaspoon);
Nonfat Milk—8 oz.

Nutrients	Calories	Protein	Calcium	Iron	Vitamin A	Thiamine	Riboflavin	Niacin equiv.	Ascorbic Acid
Totals supplied by Basic Breakfast	503	20.9 gm.	0.532 gm.	2.7 mg.	588 I.U.	0.46 mg.	0.80 mg.	7.36 mg.	65.5 mg.
Recommended Dietary Allowances—Women, 65 Years (58 kg.—128 lb.)	1800	58 gm.	0.8 gm.	12 mg.	5000 I.U.	1.0 mg.	1.5 mg.	17 mg.	70 mg.
Percentage Contributed by Basic Breakfast	27.9%	36.0%	66.5%	22.5%	11.8%	46.0%	53.3%	43.3%	93.6%

Cereal Institute, Inc.: Breakfast Source Book. Chicago: Cereal Institute, Inc., 1959.
Food & Nutrition Bd.: Recommended Dietary Allowances, Revised 1958. Natl. Acad. Sci.—Natl. Research Council Publication 589, 1958.
Watt, B. K., and Merrill, A. L.: Composition of Foods—Raw, Processed, Prepared. U.S.D.A. Agriculture Handbook No. 8, 1950.

**The allowance levels are intended to cover individual variations among most normal persons as they live in the United States under usual environmental stresses. Calorie allowances apply to individuals usually engaged in moderate physical activity. For office workers or others in sedentary occupations they are excessive. Adjustments must be made for variations in body size, age, physical activity, and environmental temperature.*

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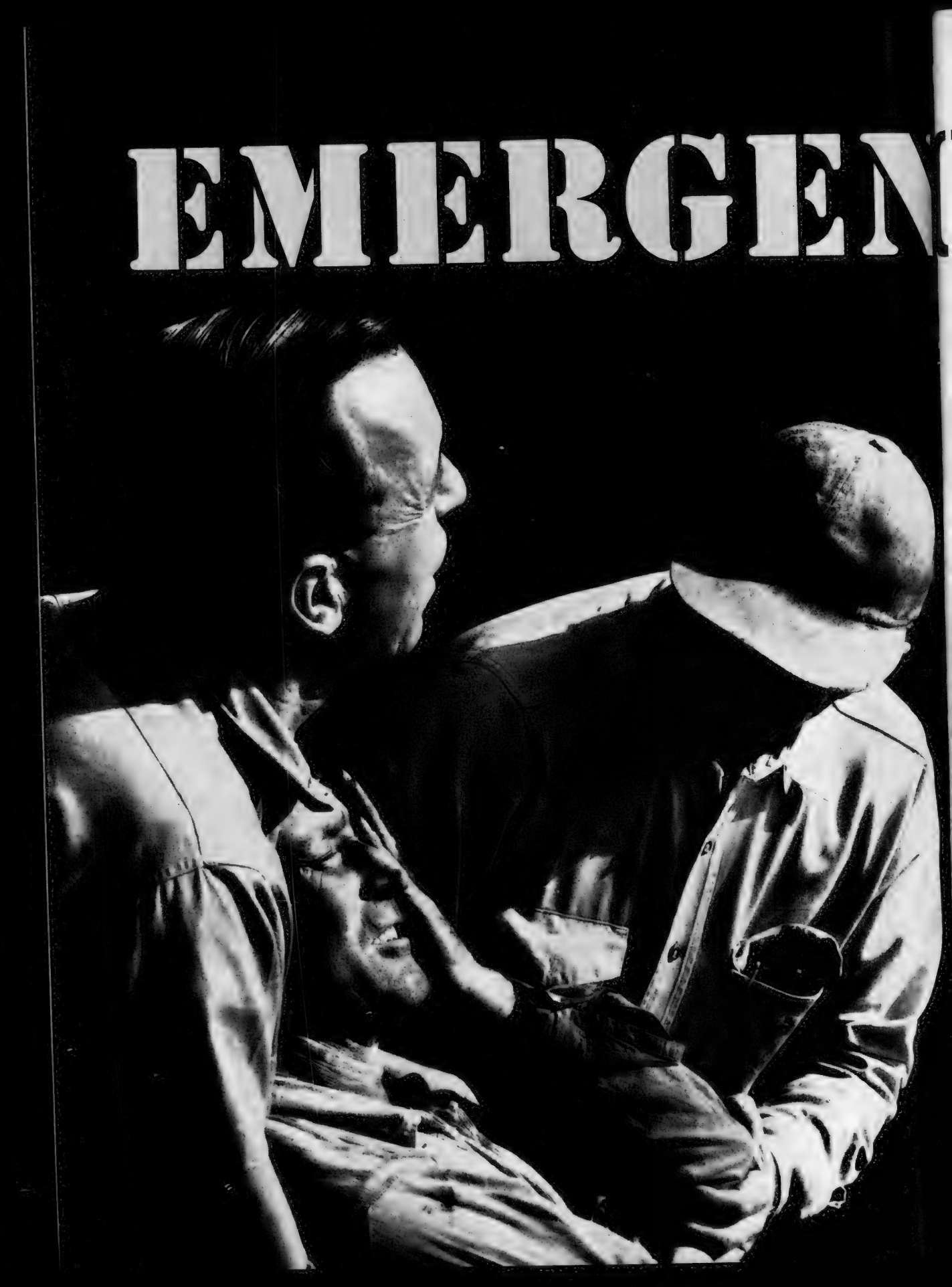
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Journal of Occupational Medicine

Tuberculosilicosis: A Study of Its Synergistic Mechanisms

Paul Gross, M.D., Marian L. Westrick, Ph.D.; and
James M. McNerney, M.P.H., Pittsburgh, Pa.

THE NATURE of the synergism between tuberculosis and silicosis has intrigued scientists for decades. Kettle's hypothesis[1] for this synergism, until recently the only one extant, assumed that tubercle bacilli had a predilection for tissue containing silica. A new hypothesis, advanced by Vigliani and Pernis,[2] suggests that the synergism is based on a nonspecific reciprocal stimulation which multiplies the respective specific immune responses. Three years ago we also began an investigation to elucidate the mechanism of this synergism.

It is the purpose of this paper to give a detailed report of this study, the results of which seem to support the concept of Vigliani and Pernis.

Materials and Methods

Guinea pigs were rendered tuberculin-positive with a subcutaneous injection of a six-week-old culture of human tubercle bacilli. This culture* was derived from the "B" strain used by Zaidi and co-workers in King's laboratory.[3] It was only weakly pathogenic for guinea pigs.

After the demonstration of positive tuberculin reactions, some of the reactive guinea pigs were given a single intratracheal injection of 50 mg of quartz dust suspended in 1 ml of saline which also contained 0.01 of PPD tuberculin. Other reactive animals were given a number of such injections at monthly intervals except that the quartz dust dosage was cut to 25 mg. By x-ray diffraction, the silica dust consisted of 84 ±6 % alpha quartz and had an average particle size of 0.7 μ (micron). It had previously been demonstrated not to produce nodular or significant collagenous lesions in the lungs of guinea pigs because of the admixture of 8%-9%.[4]

The control groups were composed of reactive guinea pigs injected intratracheally with single as well as multiple doses of the dust alone and with single as well as multiple doses of the tuberculin alone.

The intratracheal injections were accomplished per os under light ether anesthesia by means of an illuminated laryngoscopic speculum.[5] The experimental set-up is shown below.

A number of the animals died of pneumonia during the course of the experiment, and others were killed with ether at various intervals in

Experimental Set-Up of Intratracheally Injected Tuberculin-Positive Guinea Pigs

Group	No. of Guinea Pigs	Single or Multiple Dosage	Total Dosage (mg) Quartz	Tuberculin
Tuberculin with quartz dust	30	S	50	0.01
Tuberculin with quartz dust	30	M	150	0.03
Tuberculin alone	15	S	..	0.01
Tuberculin alone	15	M	..	0.03
Quartz dust alone	15	S	50	..
Quartz dust alone	15	M	150	..

From the Industrial Hygiene Foundation.

This investigation was made possible by Research Grant No. E-1710 from the National Institutes of Health, Public Health Service.

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*Obtained through the kindness of Prof. C. V. Harrison, of the Postgraduate School of Medicine, London.

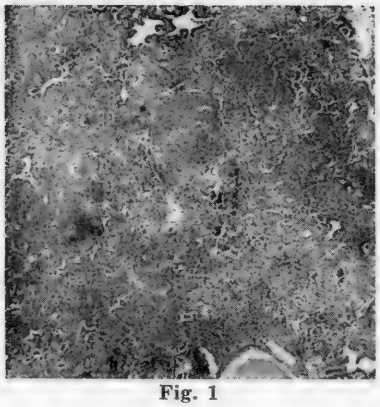

Fig. 1

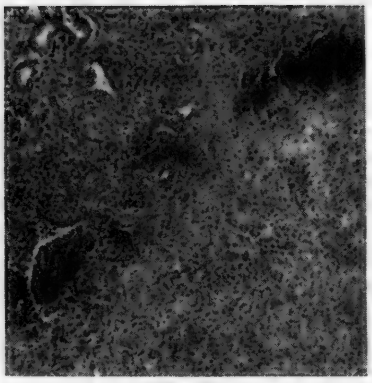

Fig. 2

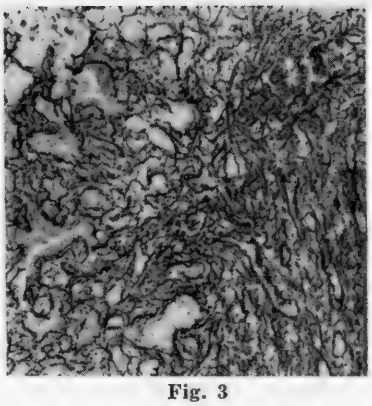

Fig. 3

Fig. 1—Extensive consolidation of lung in tuberculin-positive guinea pig injected intratracheally with 150 mg of quartz dust (nonfibrogenic) and 0.03 mg of tuberculin. This animal was killed 13 months from the initial injection. H. & E. Reduced approximately one-half from magnification ×36. Fig. 2—A higher magnification of the pulmonary consolidation in a tuberculin-positive guinea pig injected intratracheally with 50 mg of nonfibrogenic quartz dust and 0.005 mg of tuberculin. This animal died 10 months later. The consolidation is caused by a proliferation of cells with ovoid nuclei, apparently alveolar cells. There is focal necrosis with calcification. Some surviving air spaces are lined by metaplastic columnar epithelium. H. & E. Reduced approximately one-half from magnification ×60. Fig. 3—The reticulin pattern of the preceding field shows extensive obliteration of air spaces by proliferated reticulin fibers. Gordon and Sweets procedure. Reduced approximately one-half from magnification ×160.

order to study the progress of the tissue changes. The survivors were killed with ether at the end of 13 months. All animals were autopsied and the lungs fixed by injecting formalin intratracheally. Paraffin sections were prepared and stained with hematoxylin and eosin.

Selected fields on many of the sections were photographed at a magnification of ×290. The position of the fields was located by means of a Field-Finder.† After the photography, the sections were decolorized and impregnated with silver by the Gordon and Sweets method to demonstrate reticulin fibers. The same fields were then rephotographed. The sections of animals that had received quartz dust were incinerated at 600°C for one hour, and after treatment with concentrated hydrochloric acid the various fields were again rephotographed, but this time with dark-field illumination. The negative of the dark-field photograph when superimposed upon the first negative of the

†Sold by W. & L. E. Gurley, Engineering Instruments, Troy, N.Y.

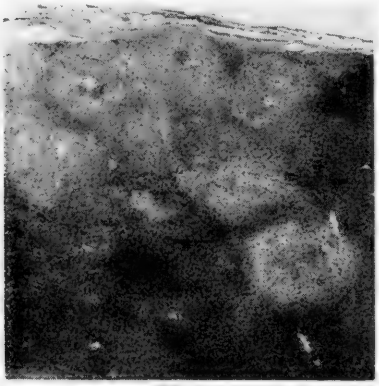

Fig. 4

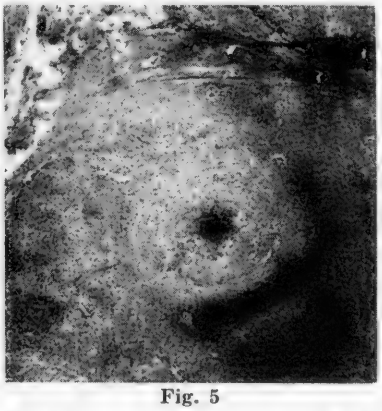

Fig. 5

Fig. 6

Fig. 4—Miliary granulomatous nodules in a paratracheal lymph node of a tuberculin-positive guinea pig injected intratracheally with 50 mg of nonfibrogenic quartz dust and 0.005 mg of tuberculin. This animal was killed nine months later. H. & E. Reduced approximately one-half from magnification ×36. Fig. 5—A higher magnication of a granulomatous nodule in a node from the same animal as in Fig. 4. The nodule has three zones: a central necrotic one, a middle zone with radially arranged fibers, and an outer zone with concentrically arranged fibers. H. & E. Reduced approximately one-half from magnification ×64. Fig. 6—The reticulin pattern of the preceding field shows the three zones in the nodule more clearly. Gordon and Sweets procedure. Reduced approximately one-half from magnification ×64.

same field constituted a record of the amount and topographic distribution of the quartz dust in that field. More than 1,800 negatives were so prepared from more than 700 fields.

Results

Experimental Group

The predominant pathologic change in the lungs resulting from intratracheal injections of quartz dust in combination with tuberculin is an extensive consolidation of air spaces by closely packed cells with ovoid or round, pale nuclei and ill-defined cell boundaries (Figs. 1 and 2). The reticulin preparations confirm the obliteration of air spaces and terminal bronchioles by a complex and irregular network of reticulin fibers (Fig. 3). In some fields this obliteration is not complete and spares an air space here and there. In some of the lungs there is focal necrosis in or near the center of such areas of obliterative alveolitis, and in several lungs the necrotic tissue is calcified. The necrosis is bland and is not accompanied by exudation of any kind. In the necrotic foci some of the reticulin fibers show fragmentation and lysis. There is also condensation of reticulin fibers resulting in heavy accentuation of septal walls that are not discernible in the routinely stained sections. Collagenization is infrequently encountered and is limited to relatively small foci. When present near bronchi, the collagenization is associated with metaplasia of alveolar lining cells to the bronchial type of epithelium to form pseudoglandular structures.

Fig. 7

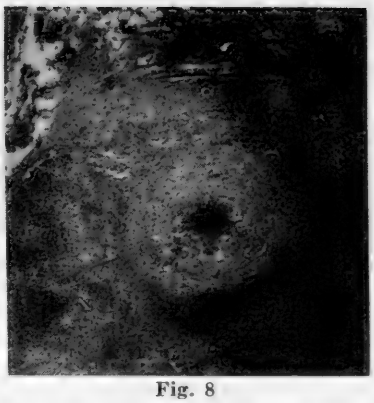

Fig. 8

Fig. 7—The composite ash picture of the field shown in Fig. 2 shows abundant storage of quartz dust except near the regions of necrosis and the pseudoglandular structures. Reduced approximately one-half from magnification ×160. Fig. 8—The composite ash picture of the field shown in Fig. 5. There is abundant quartz dust in the tissues about the nodule but extremely little dust within the nodule. In other nodules, the distribution of the quartz dust may be reversed. The disposition of the dust is unpredictable. Reduced approximately one-half from magnification ×64.

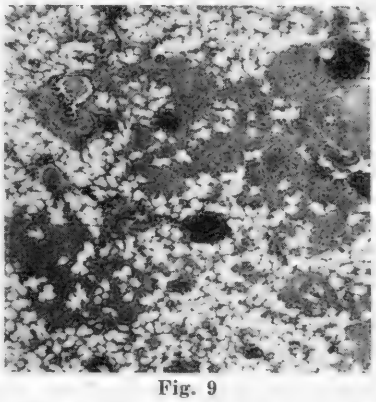

Fig. 9

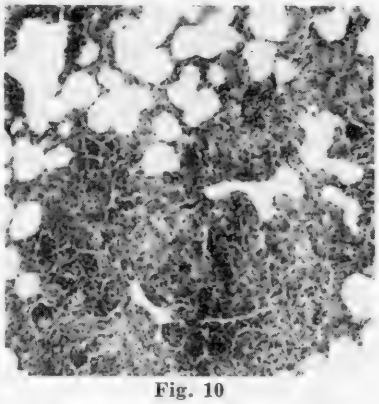

Fig. 10

Fig. 9—The characteristic lesion produced in a guinea pig lung by the intratracheal injection of 150 mg of nonfibrogenic quartz dust. This dust-control animal was killed nine and one-half months after the first injection. There is apparent consolidation, which is patchy and not extensive. H. & E. Reduced approximately one-half from magnification ×36. Fig. 10—A higher magnification of the dust-control lesion shows proliferated alveolar cells, many of which plug air spaces. Many intra-alveolar giant cells are also present. This animal received 150 mg of nonfibrogenic quartz and was killed nine and one-half months after the first injection. H. & E. Reduced approximately one-half from magnification ×160.

The paratracheal lymph nodes are usually greatly enlarged and in nearly half of the animals contain rounded granulomatous foci which, in many instances, greatly resemble miliary tubercles (Fig. 4 and 5). Some of the latter may show central necrosis, and in several animals these necrotic centers are calcified. The lymph node enlargement is due mainly to a severe hyperplasia of reticuloendothelium with associated reticulin stroma. Many Langhans giant cells may also be present. The reticulin framework of the granulomatous foci characteristically consists of three zones: (1) A central zone devoid of reticulin fibers or containing at the most only a few isolated reticulin fragments (Fig. 6). The eosin-hematoxylin stain may or may not show necrosis in this zone. (2) A middle zone of short, irregularly wavy or branching fibers that are essentially radially oriented. There may be fragmentation and lysis

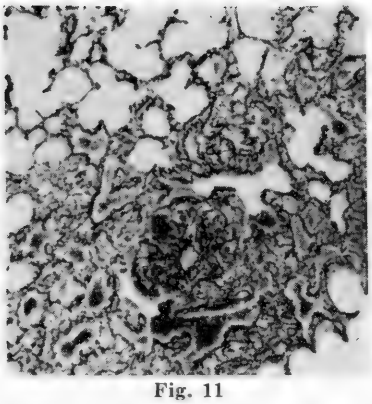

Fig. 11

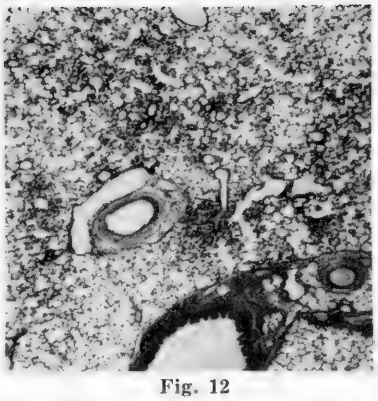

Fig. 12

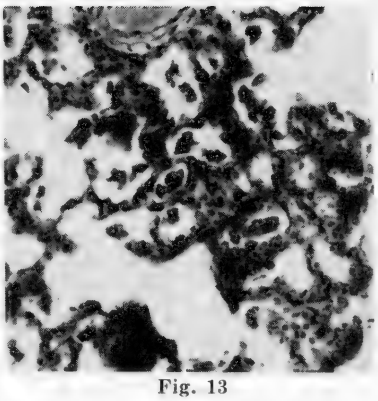

Fig. 13

Fig. 11—The reticulin pattern of the preceding field shows survival of a surprising number of alveoli. Some thickening of alveolar walls is evident, with partial obliteration of some alveoli and complete obliteration of a few. Gordon and Sweets procedure. Reduced approximately one-half from magnification ×160. Fig. 12—The persisting tuberculin lesion in the lung is characterized by patches of coarsened architecture. This is from tuberculin-positive guinea pig killed six months following an intratracheal injection of 0.005 mg of tuberculin. H. & E. Reduced approximately one-half from magnification ×36. Fig. 13—A higher magnification of the tuberculin lesion in the lung from the same animal as in Fig. 12. There is thickening of alveolar walls by proliferation of cells. Some of the air spaces contain desquamated alveolar cells and giant cells. H. & E. Reduced approximately one-half from magnification ×290.

of the central ends of these fibers. (3) A peripheral zone of circumferentially arranged fibers.

The silica content is highly variable in both pulmonary lesions and lymph node nodules (Figs. 7 and 8). In general, the mineral content of these lesions is unpredictable. Collagenized or necrotic pulmonary foci usually contain little or no demonstrable silica. However, exceptions are frequently encountered. In the lymph nodes, the granulomatous nodules usually contain only scattered flocs. In a few of the nodules, no dust was demonstrable.

Control Groups

(a) Quartz Dust—The lesions in the lungs of these animals are identical to those previously described in connection with iron-contaminated quartz dust.[6] They consist of dense accumulations of dust-filled cells within air spaces of an ill-defined, relatively small region or more extensive areas involving large portions of a lobe (Figs. 9 and 10). The alveolar walls are moderately thickened by proliferated surface cells which are supported by a network of reticulin fibers (Fig. 11). Relatively few alveoli are found obliterated by the proliferating alveolar cells. Although collagen formation is generally absent, the lungs from several of these dust-control animals show scattered small, ill-defined foci of mural collagenization. Silicotic nodules are absent. No pulmonary necrosis is encountered except in regions of suppuration from spontaneous infections.

The paratracheal lymph nodes are enlarged and are the site of diffuse reticuloendotheliosis, often associated with many giant cells. The paratracheal nodes of only three guinea pigs contain granulomatous foci which resemble miliary tubercles and which are very similar to those seen in nearly one-half of the quartz dust-plus-tuberculin animals. The lungs of these three animals are different from other lungs in the group in that they contain foci of chronic suppuration.

(b) Tuberculin—The characteristic pulmonary lesion due to tuberculin in these reactive guinea pigs has recently been described.[7] The significant features of the persisting tuberculin reaction in pulmonary tissue include a focal interstitial pneumonitis of much milder degree and greatly more limited extent than is found in lungs that have reacted to iron-contaminated quartz dust (Fig. 12). Neither the alveolar proliferation nor the reticulin fiber production tends to cause alveolar obliteration. The air spaces contain few to many giant cells and some desquamated alveolar cells (Fig. 13). In some animals, eosinophilic infiltration of the thickened alveolar walls is present. No collagen production or necrosis is found. The paratracheal lymph nodes are not enlarged and show no significant microscopic lesion, except for occasional reticuloendothelial hyperplasia and occasional Langhans giant cells,

Discussion

The basic pathologic change which underlies a tuberculopneumoconiotic process, inclusive of tuberculosilicosis, is a diffuse obliterative alveolitis.[6] Necrosis and cavitation are secondary features which may not always be prominent

and may even be absent. Crystalline silica also causes obliterative alveolitis, but generally as sharply delimited foci in the form of silicotic nodules. Iron-contaminated quartz dust, however, is capable of causing only minimal or negligible obliterative alveolitis; and tuberculin, in the doses employed, is incapable of causing any alveolar obliteration.

The combination of the iron-contaminated quartz dust with tuberculin has produced a diffuse obliterative alveolitis that is indistinguishable from the recently described experimental tuberculosilicosis[6] for the production of which living tubercle bacilli were employed. In other words, these results suggest that in a sensitized animal tuberculin may reinforce and exaggerate the reaction of pulmonary parenchyma to quartz dust in a manner which is very similar to the action of living tubercle bacilli upon silicotic lung tissue.

Since granulomatous foci were found in the paratracheal nodes of 10% of the silica-control animals and since none of the tuberculin-control animals manifested a granulomatous reaction, it seems fair to assume that the lymph node granulomata are a tissue response primarily to the quartz dust rather than to the tuberculin. Inasmuch as the incidence of lymph node granulomata in the quartz dust-plus-tuberculin group is nearly five times that in the silica-control group, additional support is given the concept that tuberculin may reinforce and exaggerate a silicotic tissue reaction in sensitized animals.

The effect of the silica dust upon the pulmonary tuberculin reaction is more difficult to evaluate. However, the occurrence of focal necrosis, particularly in pulmonary tissue, would appear to be more characteristic of an exaggerated tuberculin reaction than of a silicotic reaction. Until recently the reaction of pulmonary tissue to tuberculin, similar to the dermal tuberculin reaction, was considered to be evanescent, subsiding after 48 hours and being nearly completely resolved after one week. The recent report[7] describing the persistence of the pulmonary changes due to tuberculin reaction for months after its initiation makes it advisable to reassess the role which tuberculin reactivity may play in pulmonary tuberculous infections as well as in tuberculosilicosis.

Inasmuch as the inflammatory reaction due to silica dust requires many months for its full development in the lung, the increased severity of such dust lesions in animals which had received concomitant tuberculin gives additional support to the concept of a persisting tuberculin reaction in the lungs. The fact that silica dust plus tuberculin resulted in a greater incidence of nodular granulomatous lesions in satellite lymph nodes than was caused by silica dust alone suggests that a portion of the injected tuberculin was not fixed to the pulmonary tissue. Enough of the tuberculin must have been circulating free some months after its introduction into the lungs to produce metastatic lymph node lesions in conjunction with the stored silica dust.

Conclusions

Iron-contaminated quartz dust, when injected intratracheally in combination with tuberculin into sensitized guinea pigs, produced a greatly exaggerated tissue response. In many of the animals, the resulting disease resembled experimental tuberculosilicosis.

The modification of experimental silicosis by an immune reaction, as exemplified by the pulmonary tuberculin reaction, supports the hypothesis that the development of tuberculosilicosis is dependent upon a reciprocal stimulation of tissue responses caused respectively by silica dust, on the one hand, and by sensitivity to the tubercle bacillus and its products, on the other, as postulated by Vigliani and Pernis.[2]

(The authors' address is Industrial Hygiene Foundation, 4400 Fifth Ave., Pittsburgh 13, Pa.)

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Mental Health and the Worker

Stanley Greenhill, M.D., Edmonton, Alberta

HUMAN factors affecting mental health and productivity are to be found in three main areas of human relationships—the family, society, and the place of work. Some understanding of the human factors involved in each of these three areas is essential if an intelligent interpretation of change in work productivity is desired. A review of the significant factors involved in each of these settings is, therefore, in order. The actual manifestations of impaired productivity at the place of work may then be discussed with some real understanding.

An employee cannot be split up into attitudes and aptitudes; he is an individual. If this be the case, then anybody designated an "employee" is subject to the same trials and tribulations as the rest of mankind. It should be remembered that "employees" far outnumber the self-employed in this day and age. To be an "employee" is the rule, not the exception.

If an employee is not a peculiar subspecies of the human race, but a representative example of it, it is appropriate to speak in generalities of the formative factors and influences that eventually make us as we are. Each of us is as individual as our fingerprints. Humans do not as yet come off assembly lines.

The Emotional Development of the Individual

Our life history still starts at birth. This precipitous entry into a troubled world is, in itself, sufficient to produce ill effects in later life! However, the indignity of being born is compensated for by maternal affection. The comfort and warmth of the womb is replaced by the fondling, love, and food of the mother. The newborn's feelings of insecurity are soon replaced by feelings of security provided by this maternal affection.

During the very early years, a child tends to be egocentric and comparatively unaware of its external environment, though aware in some intuitive way of the emotional atmosphere surrounding it. In our culture, the mother is the dominant love figure. Soon the child becomes aware of his external environment and its other dominating figure, the father—the symbol of authority either benevolent or harsh. The child also becomes aware of brothers and sisters.

Gradually, he is made to realize he is a member of a social unit—the family—and that this unit is constantly making demands on him. In consequence, he has to learn to adapt. This adaptive process is not easy. It is characterized, as far as the child is concerned, by gross inconsistency in the behavior of those around him. At times he is petted, coddled, or rewarded for his efforts. On other occasions he meets with verbal and even physical abuse.

If the child experiences growing pains as he develops within the unity of his family, he experiences further pains as he becomes aware of the society existing outside the family circle. The beliefs and attitudes acquired from his family do not always coincide with the beliefs and attitudes he meets outside. To resolve these opposing views is a trying experience! For the first time he is called upon to make decisions and formulate his own ideas. Quite often these are at variance with those of his family.

As our potential employee moves from puberty to adolescence, further trials and tribulations face him. If he has managed up to now to cope with the social problems of family life and adapt to society outside, he now is faced with the bigger problems of his emerging adulthood. The adolescent, anxious to cut the family's strings, realizes with some bitterness that he is still in many ways a fledgling. In consequence, adolescence is characterized by a revolt against authority with some feeble attempts to assert individuality. This phase of emotional development is usually stormy. However, most adolescents manage to survive and reach adulthood.

Adulthood should be characterized by development of maturity. Maturity has been reached when an individual acquires some consistency of viewpoint with regard to life, and

Based on an address given to the Third Occupational Health Conference, Vancouver, B.C., Feb. 27, 1960.

Dr. Greenhill is Professor and Head of the Department of Social and Preventive Medicine, University of Alberta.

some degree of emotional stability and control in dealing with its vicissitudes. Obviously maturity cannot be equated with chronological age.

The Social Milieu

Only passing reference has been made to the part played by the social milieu in the emotional development of our potential employee. Emotional development cannot occur in a social vacuum. To attempt to understand emotional development without attempting to understand some aspects of contemporary society would be inconsistent with the holistic approach of psychosocial medicine.

Life in today's society is characterized by its fragmentation. This fragmentation is most apparent in the industrialized countries. However, the speed of development in the so-called underdeveloped countries is now proceeding at such a pace that manifestations of social stress and strain are already apparent in them. Life today, for most of us, is a many-faceted existence. Our lives are split between home and job, work and leisure.

Society in more primitive countries is much less complex. Life in a primitive society has a certain cohesiveness. This sense of cohesion is lacking in highly urbanized and industrialized civilization. In the so-called simpler communities or societies, every individual understands and participates in the various economic and social activities of the group. This understanding and participation enhance this cohesiveness and link all members into an intimate whole. In our frenzied society, there is little connection between a man's working and domestic lives. Our social environment continues to change at an amazing speed. These changes result in individuals' no longer being aware of their function and role. In consequence, they suffer from confusion and conflict. Primitive societies, on the other hand, maintain their stability because of their slow rate of change.

The emotional development of a child born into a primitive society is markedly different from that of one born into an urbanized and industrialized society. A child in a primitive society is soon made aware of his position and function. The tribe or clan into which he is born has, in itself, certain mothering characteristics. This child does not suffer from social loneliness, rejection, or feeling of not belonging. The speed of social change in our society, with its resultant loneliness, rejection, and feelings of not-belonging, undoubtedly contributes greatly to the rising incidence of nonorganic disease.

Some Changes in Social Attitudes

Perhaps changes in attitudes toward marriage are the most striking. The trend to earlier marriages is resulting in significant sociological changes. Couples are not only marrying earlier but producing children earlier and controlling family size. In consequence, the average young couple produces 2.8 children before the mother has reached the age of 26. This fact has started a sociological chain reaction.

Most probably this desire for house and home and family by today's young adults is an expression of their unconscious yearnings for security in a world which offers less and less security with the threats inherent in every new technological achievement. In this rootless, unstable, threatened society, it is perhaps natural that young couples should seek the solace and security offered by the most primitive of human groups, the family.

However, this desire for security also exhibits some of the social maladjustments typical of the mid-twentieth century. These young couples are not self-sufficient units. Though anxious to be secure, they show little desire to struggle for that security. Though eager to have house and home, they wish it to be given to them on a platter. The material things, assumed to be so necessary for a full life, are wanted immediately. The gradual acquisition of belongings is too slow a process in this uncertain age. As a result, the average young couple of today is in a state of financial indebtedness that would have been considered intolerable by their parents or grandparents. Many young couples look as though they will celebrate the end of their indebtedness about the time they receive their first old-age-pension check! Because of their financial state, today's young couple finds it more essential than ever to maintain their wage earning potentials at as high a level as possible. The young married employee, with family, mortgaged house, and unpaid for "essentials," naturally seeks employment that offers the most security. Ambition and adventure are of secondary importance. The main thing is to find something that is sure, a job that pays well, a job with an assured annual vacation and a wide variety of fringe benefits. A distant Eldorado does not appeal.

The wife also has similar ideas. Unlike previous generations, the wife of today, by the time she reaches 30, has fulfilled her obligation as a procreator of the race. She is now in a position to help fill the family coffers. Education- and opportunity-wise, today's young matron has a far better chance of becoming an economic self-sufficient unit than ever before.

This refilling of the coffers at increased rate by having the wife work is undoubtedly an attractive proposition. It is also an anxiety-producing situation. Feelings of guilt, doubt, and uncertainty arise in the working mother, as she contemplates the potential harmful effect her absence may have on the children, feelings that are not lessened by the free psychiatric advice offered by press, radio, television, magazines, and well-wishing but poorly informed friends. This inner turmoil is bound to be reflected in the atmosphere of the home and manifested in the children by periodic crises in their emotional development.

Today's family does not have the stability of yesteryears. We are tending to become a population of migrants. In the United States, 20% of families change their place of residence annually. This change in residence is not only a trek from rural to urban areas, but also a move from urban to suburban areas. The stress and strain of suburban living has been the source of plot and income for many writers in recent years. Even when allowance is made for overemphasis on the part of such authors, it is nevertheless true that rootlessness, financial indebtedness, absence of parental authority, and frenzied seeking for status are major contributors to today's social pathology.

The Work Situation

The place of work bears a close resemblance to the environment in which the employee grew up. A large organization has many of the attributes of the mother figure. Love and affection have been converted to pay checks, holiday with pay, and fringe benefits. Identification of supervisors and foremen with the archtype father figure is common. Rivalry and competition with one's fellow workers is remarkably reminiscent of relations with brothers and sisters. Mental attitudes resulting in inefficiency at work can nearly always be traced back to the formative years of those involved in a work situation problem.

Psychodynamics of the Work Place

The place of work becomes the stage on which can be observed the interplay of emotional forces. The home, parents, siblings, and society on the one side; the organization, management, supervisors, and fellow workers, on the other. On this stage are reenacted the same emotion-producing situations as occurred at an earlier age. The passage of time and the changed social setting, however, have modified them. No longer do these emotions show the lability of youth; instead they demonstrate the rigidity of age—in other words, youthful emotions have become adult attitudes. Human work problems are but a passing scene in front of this stage setting. Rarely does a glance at a scene tell us the whole story!

Hence the emphasis that has been placed upon the individual's emotional and social development. It is too simple to explain or blame an unpleasant work incident on some superficial remark made by a fellow-worker, a mild criticism by a supervisor, or a minimal wage adjustment by management. The true significance of this unexpected reaction can be appreciated only if there is some awareness of the psychodynamics involved.

Latent attitudes to parental authority may become overt in the presence of a supervisor. Deep-seated family prejudices may reemerge at the sound of a foreign accent. Long-forgotten childhood rivalries may reemerge in the struggle for promotion. A mild reprimand may result in a prolonged depression.

A comprehensive objective approach, such as has been outlined, demands knowledge and understanding of ourselves. We all tend to view our fellows through the distorting lenses of our biases, prejudices, and preconceived ideas. Our assessments of human behavior problems tend to take on the characteristics of a moral judgment. Emotional neutrality is essential if our efforts at handling the problems of others are to be successful.

Conclusion

Most human factors affecting mental health and productivity will not be found clustered around the work place. They will, instead, be found in diverse and unsuspected spots along the road from infancy to adulthood. A few hints are given as to the most likely spots where these factors may be found.

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Air Quality for California Communities

Malcolm H. Merrill, M.D., Berkeley, Calif.

THE HARMFUL effects of air pollution can be both widespread and varied. They are physical, biological, psychological, aesthetic, and economic in nature. Man, animals, plants, and a variety of inanimate things are subject to its unwelcomed effects. It is because of these effects that there is concern for air pollution. There is a growing awareness of decreased visibility, damage to vegetation, deterioration of materials, and discomfort to human beings in various degrees.

Air pollution in its most acute and dramatic form was evidenced in the disastrous episodes which took place in Donora, Pa., in the Meuse Valley in Belgium and in London, England. In each of these instances, loss of life was related to a severe attack of air pollution. The effects of air pollution are observed in the highly uncomfortable smog of Los Angeles and, to a lesser extent thus far, in other areas of California where the adverse effects on the comfort of citizens, the transparence of the atmosphere, and the health of plant life are also evident.

Although air pollution is of concern to agriculture, real estate, transportation, and many other interests, our most important concern, in the last analysis, is the effect on people.

Aside from the knowledge that air pollution has resulted in death to a large number of persons during the Donora, Meuse Valley, and London episodes, it is known that it also interferes with the comfort and well-being of entire communities. A health survey by the California Department of Public Health in 1956 and 1957 showed that two-thirds of the people living in Los Angeles were bothered by air pollution either at home or at work or both. Elsewhere in California where air pollution was not yet considered to be as serious a problem, almost one-third of those interviewed answered they were bothered.

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The prevention of interference with comfort and enjoyment of life alone is of sufficient significance to justify control programs directed toward maintaining air quality at satisfactory levels.

Here in California and particularly in Los Angeles, air pollution has become one of the most important problems facing our metropolitan regions. In many ways, it is suprising that a problem as serious as that now present in Los Angeles was first recognized only some 15 years ago. During this period, however, the people of the state have come to realize that the air supply of our communities is limited and that it can be seriously impaired by the activities associated with community life. As a result, there is strong public support and even demand for action on the part of local and state agencies to deal with air pollution.

The urgent need to solve this problem can be appreciated if the present levels and extent of air pollution in California are considered together with the growth that is expected to take place. The counties of the state in which communitywide air pollution has been experienced comprise almost 80% of California's 15,000,000 citizens. These counties as well as most of the others in the state are growing at a rapid rate, and the state population is expected to double within the next 25 years. It is obvious that areas where air pollution now exists will have even more frequent and severe attacks unless effective control measures are undertaken. Other communities not yet experiencing air pollution will be faced with this problem as they become more densely populated.

As one step in the effort to cope with the air pollution problem, the 1959 state legislature passed a bill that requires the State Department of Public Health to adopt and publish standards for air quality. This bill, which was signed by Governor Edmund G. Brown, states in part: "The State Department of Public Health shall, before February 1, 1960, develop and publish

standards for the quality of the air of this State. The standards shall be so developed as to reflect the relationship between the intensity and composition of air pollution and the health, illness, including irritation to the senses, and death of human beings, as well as damage to vegetation and interference with visibility."

The Act also provides that the standards may be amended from time to time and that they shall be adopted only after public hearings have been held.

Because of the interest of this group with air quality in workroom areas and your knowledge of standards for industrial workers, you will wish to know what the Department of Public Health is doing in the area of standards for community air quality for the general population.

The most important concern with the air in our communities is that it be safe to breathe, that it not cause discomfort to people or interfere with their enjoyment of life, and that it not produce unreasonable economic loss. Levels at which each of the above effects are experienced will vary with many factors; some of the most important are the nature of the polluting substance, duration of exposure, and sensitivity of receptors, whether human or botanical. With the recognition of the variety of emission sources, it is clear that air-pollution-abatement efforts will be costly and some minimum of pollution will be unavoidable in metropolitan areas. It then becomes important to estimate what sort of pollution may be present without producing adverse effects on health or unreasonable economic loss.

There is considerable precedence for air quality standards. In dealing with complex problems involving the welfare and safety of people, it has been found advantageous to adopt standards in many other fields. A very common one is the speed limit on highways and city streets. Others include standards for electrical wiring and plumbing in buildings, foods and drugs, and licensing in professional fields. In order to control our environment, it has been found necessary to adopt standards on the chemical and bacterial quality of drinking water, bacterial quality of milk and shellfish, radioactivity, sewage disposal, and the quality of the air for workers in industry. All these standards have served a very useful purpose in providing guidelines for planning and carrying out environmental sanitation programs to protect the public health.

To date, very little has been done in this country on standards for the air in communities. The Air Pollution Authority of the State of Oregon made a start when it established levels for particle fallout and suspended particulate matter. Table I shows the levels that were set by that Authority and indicates how these standards provide for different values in industrial and residential areas.

Table I—Permitted Particle Fallout Rate and Suspended Particulate Matter in Excess of Normal Background Values. Orgeon Air Pollution Authority.

	Residential or Commercial Land Use Area	Industrial Land Use Area
Particle Fallout Rate	15 tons per sq mi per mo	45 tons per sq mi per mo
Suspended Particulate Matter	150 μg per M^3	250 μg per M^3

Table II—Alert Stages for Toxic Air Pollutants. Los Angeles County. (ppm)

	First Alert*	Second Alert†	Third Alert‡
Carbon Monoxide	100	200	300
Nitrogen Oxides	3	5	10
Sulfur Oxides	3	5	10
Ozone	0.5	1.0	1.5

* This is a close approach to the maximum allowable concentration for the population at large—still safe but approaching a point where preventive action is required.

† This is the air-contamination level at which a health menace exists in a preliminary stage.

‡ This is the air-contamination level at which a dangerous health menace exists.

Recently, the council of the Academy of Medicine of Cincinnati adopted an air pollution report[1] in which standards were recommended for SO_2, H_2SO_4 mists, and ozone. The council recommended the following maximum concentrations: ozone or total oxidants—0.1 ppm, with a limit of 0.05 for a daily average; SO_2—0.2 ppm for an eight-hour exposure; and H_2SO_4 mist—0.3 mg per cubic meter for a single-day average.

Concentrations for four substances—ozone, oxides of nitrogen, sulfur dioxide, and carbon monoxide—have been included in the rules and regulations of the Los Angeles County Air Pollution Control District as part of an alert system. These concentrations were not intended to include all the effects that will be considered in the standards to be adopted by the State Board of Public Health but were established for the purpose of preventing a possible disaster in Los Angeles County. The concentrations of these substances for the three alert levels are shown in Table II.

The Russians have been the only ones to set atmospheric standards for a large number of

compounds. Recent translations of the Russian literature containing these standards have appeared in technical journals within this country. In general, the levels adopted by the Russians appear to be very low when considered in light of our knowledge of the toxicity of many of the substances included. Further study and evaluation of the Russian literature is needed to determine if their findings are adaptable to our situation.

One other ambient air standard that should be mentioned is that for beryllium. The Atomic Energy Commission recommends that the concentration of beryllium in the community atmosphere be below 1/100 μg per cu m of air. This value is 1/200 of its recommended level for the worker exposed to this material at his place of work.

The largest volume of data available in the United States on air quality and its effects on individuals is that which has been developed in the field of occupational health and which is concerned with the protection of the industrial worker. You are certainly aware of the threshold limit values for a long list of compounds that have been adopted by the American Conference of Governmental Industrial Hygienists. These levels have been determined with use of data developed by several methods. For example, some of the levels were based on a combination of animal experimentation and experience with workers under actual working conditions; others were developed largely on animal experimentation alone; and still others were based on judgments formed by observing sensory responses of persons.

It would seem that perhaps the concentration of substances used to protect the health of workers could be applied directly to the atmosphere of a community. Unfortunately, this is not the case. Worker exposure is for an eight-hour day and five-day week; industrial workers are not representative of the most sensitive or frail groups in our population. Furthermore, levels for industry are based on exposure to single substances. Consequently, the nature of exposure to people at work is considerably different from that of populations subjected to a complex type of air pollution such as that occurring in California metropolitan regions. In fact, the American Conference of Governmental Industrial Hygienists recognized these difficulties when, in adopting the threshold limit values, it stated, "They are not intended for use, or for modifications for use, in the evaluation or control of community air pollution or air pollution nuisances."[2]

Even though the industrial levels cannot be applied directly to the outdoor atmosphere, they will be most useful in our efforts to adopt ambient air standards. This is particularly true when the industrial levels are based on data from human experience or animal experimentation. Table III compares maximum allowable concentrations for industrial workers with maximal and average concentrations of a number of compounds in polluted atmospheres in California. While these are not shown to in-

Table III—Comparison of Industrial Threshold Limits, M.A.C. (U.S.), Maximal Air Pollution Values (California), and Average Eight-Hour Values for Days with Severe Air Pollution (California)

Substance	United States: Industrial Threshold Limits "M.A.C."* ppm	United States: Industrial Threshold Limits "M.A.C."* mg per M³	California: Maximal Air Pollution Levels†	California: Average Eight-Hour Values for Days with Severe Air Pollution‡
Carbon monoxide	100	100	88 ppm	20-25 ppm
Fluorine	0.1	0.2	0.03 ppm	..
Formaldehyde	5	6	1.87 ppm (aldehydes as formaldehyde)	0.2-0.8 ppm (aldehydes as formaldehyde)
Gasoline	500	2,000	4.66 ppm (total hydrocarbons)	0.2-0.4 ppm (total hydrocarbons)
Heptane (*n*-heptane)	500	2,000		
Hexane	500	1,800		
Hydrogen sulfide	20	30	0.9 ppm	..
Nitrogen dioxide	5	9	1.74 ppm ($NO+NO^2$)	0.1-0-2 ppm ($NO+NO^2$)
Ozone	0.1	0.2	0.90 ppm	0.15-0.2 ppm
Sulfur dioxide	5	13	2.49 ppm	0.1-0.3 ppm
Arsenic	..	0.5	0.00065 mg/M³	0.0004 mg/M³
Lead	..	0.2	0.042 mg/M³	0.006-0.015 mg/M³
Vanadium—dust	..	0.5	0.00017 mg/M³	..
—fume	..	0.1		

*Threshold Limit Values for 1958, *A.M.A. Arch. Indust. Health* 18:178, 1959.

†Data furnished by the Los Angeles County Air Pollution Control District or obtained from Stanford Research Institute. Literature Review of Metropolitan Air Pollutant Concentrations. Preparation, Sampling, and Assay of Synethetic Atmospheres. 1956.

‡Estimated by California State Department of Public Health, based on monitoring data.

dicate what standards this department will adopt, the comparison will be of interest to those who are acquainted with the threshold limit values.

Only for ozone does the measured average eight-hour air pollution level exceed the industrial threshold limit. For carbon monoxide, the maximum air pollution value is near the threshold level and the eight-hour level is about one-fourth the threshold level. For lead, sulfur dioxide, and nitrogen dioxide, the value is about one-twentieth. Attention is directed to the fact that air pollution monitoring does not always measure a substance for which there is a threshold level. Hence, the formaldehyde threshold value is not strictly comparable to "aldehydes as formaldehyde" in community air. Nor is nitrogen dioxide comparable to $NO+NO^2$.

Other information will also be used by the department in setting the atmospheric standards. Data are available on air pollution levels over several years at a number of locations in the state. Information is also available on the occurrence of irritation to the eyes and respiratory system and damage to vegetation in several of the metropolitan regions where air pollution is a problem. Vegetation damage and eye irritation have been produced and observed in laboratory experiments. The Department of Public Health has conducted a number of epidemiological studies of population exposed to air pollution and supported studies on the health effects of air pollution.

Because the Act giving the responsibility for adopting standards to the department states that the standards shall "reflect the relationship between the intensity and composition of air pollution and the health, illness, including irritation to the senses, and death of human beings, as well as damage to vegetation and interference with visibility," it is apparent that one level cannot define all these conditions.

It is the present intent of the department to establish three levels to cover the broad conditions defined in the Act.

Acute sickness and death have not so far been demonstrated to have resulted from air pollution in California. Such concentrations, however, have been reached in other places. It is necessary, therefore, that levels of pollutants or combinations of pollutants likely to lead to acute sickness or death for a sensitive group be established. This could be considered to be an "emergency" level requiring prompt and drastic action.

Another level will probably be adopted for concentrations of air pollutants or possible combination of pollutants which is likely to lead to insidious or chronic disease or to alteration of important bodily function in a sensitive group. By important bodily function is meant a function such as ventilation of the lung, transport of blood gases by hemoglobin, or dark adaptation. This level would be below the "emergency" level but would represent "serious" levels of air pollution requiring vigorous and unremitting control effort. It may be that air pollution causing widespread economic loss should also be considered in our standards at this level.

The standards also will consider a third and lower level at which discomfort from air pollution is first felt, there is interference with the enjoyment of life, or economic loss results. This third level would represent concentration of pollutants below which it would be desirable to maintain the atmosphere in communities. Although air pollution control programs should be aimed at keeping air quality below this level, the urgency of adequate control measures would not be as great as for the other two levels.

Table IV indicates the form the standards are expected to take.

The pollutants in Table IV are merely used as examples of how the levels would be listed. A compound such as sulfur dioxide could appear at all three levels, since it is capable of causing crop damage at the lowest level, in higher concentration would be serious from the standpoint of human exposure, and in even still higher concentration could cause death. Carbon monoxide, on the other hand, would probably not be included at Level I, since it normally would not be considered to produce effects defined at this level. It would appear at Levels II and III, since it can interfere with important bodily function and cause death. The third example, ethylene, can cause damage to certain crops in very small concentrations and would

Table IV—Expected Pattern for Ambient Air Standards in California

Pollutants	Level I (level at which there would be beginning interference with comfort, damage to vegetation, reduction in visibility, and similar effects)	Level II (level at which concentrations of pollutants are likely to lead to insidious or chronic disease or to alteration of bodily function)	Level III (level at which concentrations of pollutants are likely to lead to acute sickness or death)
SO^2	×*	×	×
CO	—†	×	×
Ethylene	×	—	—
Others	×	×	×

* × indicates that standard would be set at this level.
† — indicates that standard would not be set at this level.

have to be considered in Level I. Its concentrations in the atmosphere, however, have been so low that it is not considered to be important to human exposure and might not be included under Levels II and III.

Since the first standards are to be adopted by Feb. 1, 1960, they must be based on information now available. It is evident that standards cannot be adopted for all compounds that are present in the atmosphere. The extension of the standards to new compounds and refinement of the levels first adopted can only be done as additional information becomes available.

The procedure for adopting the standards will include the accumulation and evaluation of existing information, the critical review of all data and the proposed standards by responsible scientists through informal public hearings, and, finally, formal hearings and action by the State Board of Public Health. The State Department of Public Health has an Air Sanitation Advisory Committee composed of persons who have had wide experience in air pollution or a related field. These people represent air pollution control districts, medicine, universities, industry, city planning, research interests, and agriculture. This committee will be used to advise us in all steps of the standard-setting. Frequent committee meetings have been held and are scheduled. The department has also selected a technical group composed of persons with experience in such fields as toxicology, medicine, and air pollution to provide information to the department and to our Air Sanitation Advisory Committee regarding specific pollutants to be considered and their concentrations. The department will also seek the advice and assistance of experts from other parts of the country, the Public Health Service of the Department of Health, Education, and Welfare, universities, research institutions, and any other sources which will be of help.

The adoption of air quality standards in California, in themselves, will not solve the air pollution problem. This can only be achieved by reducing the amount of wastes discharged into the atmosphere. The standards, however, will define the air quality that should be maintained in a community and can be used as guides for control programs. In practice, this might be similar to water pollution control. Here, standards are established for the waste-receiving waters. Effluents discharged into the receiving waters must not result in water pollution levels above those set forth in the standards.

Motor vehicles are recognized as one of the major sources of air pollution in metropolitan regions of California. The Los Angeles County Air Pollution Control District considers them to be the only major source that remains uncontrolled in that county. In addition, motor vehicles are mobile sources that move across boundaries of local air pollution control agencies. Because of the importance and somewhat unusual nature of this source of air pollution, the 1959 state legislature also passed a bill that requires the State Department of Public Health to adopt standards on exhaust contaminants from motor vehicles.

It is apparent that the motor vehicle exhaust standards are closely related to the standards for air quality. The first step is to establish the level of community air desired. Then, the degree of control of motor vehicle exhaust emissions needed to maintain the desired quality of community air must be determined.

The procedure for adopting the motor vehicle exhaust standards will be similar to that for air quality standards. Existing information will be evaluated; these data and the proposed standards will be reviewed with qualified individuals, groups, and scientists, and the standards will be considered in public hearings. The department's Air Sanitation Advisory Committee is also advising us in all the steps relating to the motor vehicle exhaust standards.

After completion of the staff work by the department and its consultants, the State Board of Public Health, after a public hearing, will take final action on the standards.

Legislation has not yet been enacted to indicate how the motor vehicle exhaust standards will be implemented. It is likely that the standards will be used as guides to establish the controls that may be required for exhaust emissions from motor vehicles.

In concluding this discussion, it is important again to point out that standards have been adopted in many other fields and have served a very useful purpose in protecting the health, welfare, and safety of people. It is hoped that the standards on air quality and motor vehicle exhausts will be of value in California's attack on air pollution.

Standards, however, are not a substitute for knowledge and good judgment. This group, more than almost any other, can appreciate the difficulty in establishing air quality and motor vehicle exhaust standards and the limitations these standards will have. It is important to point out that standards, whether they be for speed on highways, the bacterial quality of drinking water, emissions in exhausts of motor vehicles, or for air quality, are not precise figures below which there is complete safety

and above which there is certain illness, death, or accident. The standards can only act as guides. They represent levels below which it is believed nearly everyone will be protected. Obviously, the levels can sometimes be exceeded without injury to an individual. The standards that are adopted must be the products of judgment and will not represent fine lines between "safe" and "unsafe" air or between "good" and "unsatisfactory" air. They will have to be used intelligently with the appreciation of their limitations as well as their application.

Summary

The California state legislature has enacted bills requiring the Department of Public Health to establish standards for air quality and motor vehicle exhaust by Feb. 1, 1960. This paper describes the procedures that are being followed by the department in setting standards for air quality. It describes the expected pattern for the standards in California and also summarizes the efforts of others to set limits for industrial or atmospheric contaminants.

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Problems in Industrial Cardiology

F. W. Wilson, M.D., Port Arthur, Texas

THE PROBLEMS of an industrial cardiologist practicing in an industrial medical unit functioning under the code of ethics of the American Medical Association differ somewhat from those encountered in ordinary practice. His problems pertain largely to the diagnosis, functional evaluation, job placement, disposition, and consultation and the avoidance or minimizing of the snarls of litigation.

Functions of the Industrial Cardiologist

Naturally diagnosis is a basic element of the whole problem. The two greatest pitfalls here, from an industrial medical point of view, are the accuracy of previously made diagnoses and the increased medicolegal hazard of improper diagnosis. Improper diagnoses of heart disease made by persons less skilled in cardiology pose a problem to which there is no satisfactory answer. Frequently these incorrect diagnoses have been carried for years. As a result of the unfortunate class consciousness which tends to be engendered by labor-management differences, there is a tendency among many industrial workers to depreciate the opinions of any physician employed by an industry; hence any attempt by the industrial cardiologist to negate or modify such a diagnosis is apt to be met with doubt if not with actual refutation. An added psychological hazard among this group of patients is the fact that some of them have enjoyed restricted job assignments, special rest privileges, and transportation benefits and are reluctant to accept the removal of a diagnosis which could cancel these privileges. The specter which all physicians dread is the relatively rare patient who is assured that he does not have heart disease and who shortly thereafter has a heart attack. In spite of proper training and appropriate studies, this situation can and does occur. In the industrial environment, such a tragedy is far more apt to result in litigation than it is in regular practice, particularly if the patient had been told by his own physician earlier that he had heart disease, even though such a diagnosis may have been made years before the infarction.

Functional evaluation and job placement are closely related in that the job demands must be correlated with the individual's functional cardiac capabilities. This requires more than a superficial knowledge of the patient's actual duties. Job placement is no great problem for workers in sedentary jobs and varies in complexity for operating and maintenance personnel, but it presents trying problems in the rehabilitation and utilization of those who do common labor. Added problems in job assignment and reassignment are complicated by union contracts, craft lines, and the occasional case involving pay decrease which may accompany job reassignment.

Another function of the industrial cardiologist is that of consultation. It is in this field that the industrial cardiologist is most liable to find himself at odds with the regular practicing cardiologists. Heart disease per se is rarely of industrial origin, and the industrial cardiologist usually is not justified in getting involved in therapy of nonemergency nature or participating in the care of cardiacs away from the plant unless invited to do so by the patient's own cardiologist. There should be no reason for controversy in consultations performed to determine diagnosis, to aid in job placement, or to determine necessity or desirability of retirement.

It is the responsibility of the industrial cardiologist to demonstrate to the other cardiologists in the area that he is cooperating sincerely with them, that he is not using his industrial affiliation to build a private practice, and that his actions do not interfere with the economics of their practice. When these are accomplished, the industrial cardiologist usually finds that the other cardiologists welcome him in the area, cooperate with him by providing information, and freely utilize his opinions in their joint problems.

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Medicolegal Problems

The foregoing problems tend to appear simple when one enters into the complexities of the medicolegal aspects of heart disease, particularly as covered by the workmen's compensation laws. The enormity of this problem is exemplified in New York State, where an average of more than $4,000,000 is paid every year for compensation in heart case claims.[10] States recently industrialized tend to have less stringent workmen's compensation laws, but it is only a matter of time until they also become more "liberal."

From a medicolegal point of view there are six categories of heart disease. The most important of these pertains to the relationship of myocardial infarction to the stresses and strains of one's job. Master[1] and many others have emphasized that in most cases myocardial infarction occurs during sleep or other types of rest and that in only 2% of the cases are the attacks noted during or shortly after unusual strain. Master[2] further states that effort may precipitate myocardial infarction without coronary occlusion, and coronary occlusion can occur only in the face of antecedent atherosclerosis. Bell[3] and Willius[4] express the opinion that no amount of effort or physical strain can harm a normal heart. Plotz[5] points out that in New York and New Jersey unusual exertion is required for a myocardial infarction to be considered of industrial origin. In Pennsylvania[5] the claimant who was in apparent good health and sustains a myocardial infarct in the course of routine exertion is assumed to have had an accident; however, if he had known previous heart disease, unusual strain must be shown to prove an accident. In California, Iowa, and Massachusetts[5] the issue is whether or not there is fundamental causation rather than accident. There must be unexpected result and some exertion which would cause the collapse. Perhaps the greatest problem of all in determining whether a given myocardial infarction is considered to be industrially related or not is the variance of opinions of cardiologists in general. This has been demonstrated by Sigler[6-9] and his committee, as well as by Pinto,[10] Sparkman,[12] and others.

The second category is that of rheumatic heart disease. The relationship here can best be summarized by quoting from Texon's[11] excellent book: "It is generally agreed that rheumatic heart disease is not ordinarily related to trauma. It is essential to recognize the natural history and course of rheumatic heart disease in order to evaluate properly the effect of trauma in a given case. Exertion may precipitate congestive heart failure in the course of rheumatic heart disease, but exertion cannot influence significantly the pathology inherent in the rheumatic process." This statement tends to apply also to other types of valvular heart disease and to congenital heart disease.

Hypertensive cardiovascular disease tends to have a regular progression and to have its early episodes of failure at night; so it usually presents no peculiar problem in relation to workmen's compensation laws[10, 11] unless it is complicated by occlusive vascular disease, and most of these cannot be shown to be aggravated by industrial employment.

The fourth category, that of penetrating wounds of the heart, is clearly industrial in etiology; and the chief problem presented to the industrial cardiologist is early recognition of the condition so that prompt therapy may be instituted.

The fifth category is heart damage due to nonpenetrating trauma of the chest. Master[13] states that a blow against the chest or abdomen may produce contusion of the heart or functional derangement or both; however, he does not believe that contusion of the heart usually can precipitate coronary thrombosis unless the coronary artery is directly involved in the bruise. Moritz[14] opines that the diagnosis of traumatic myocardial infarction is justified only if the thrombosed vessel is in the area of contusion. The same opinion is expressed by Texon.[11]

The last category is that of the relationship of heart disease to remote trauma. Sigler[15] analyzed a series of severe injury cases and reported that in 76% there was demonstrable evidence of some cardiac damage including precordial pain, abnormal heart sounds, gallop rhythm, friction rub, systolic murmurs, or nonspecific electrocardiographic changes. Complete recovery usually is the rule. Texon[11] emphasizes that many persons with heart disease or cardiac neurosis try to blame almost any type of injury for their cardiac problems and that the majority of such charges are completely unjustified.

There are certain actions which the industrial cardiologist may take to minimize the number of injury suits, deny workmen's compensation in unworthy cases, and yet try to see that legitimately incurred occupationally injured heart cases are properly compensated. These include the following:

1. The cardiac patient should be impressed of one's sincerity and that the handling of his case will be unbiased.

2. Patients with acute myocardial infarction reporting to the industrial cardiologist at the plant should be handled promptly and efficiently, must be provided all indicated emergency therapy, and should be personally escorted, when feasible, from the plant to the family physician. There is rarely a time when a patient is more gratified to have a physician at his side.

3. Borderline cases, such as those of questionable infarction, questionable coronary insufficiency, and angina pectoris which is slowly or incompletely relieved should be sent in to the care of the family physician. Conservatism should be emphasized here.

4. Autopsies should be urged in cases of patients dying at the plant of apparent or suspected heart attacks. Such data are invaluable, particularly in delayed suits.

5. The industrial cardiologist should encourage the formation of an expert panel plan[10] in his area. Such has been used with good results in Utah for the past 10 years. This panel is composed of specialists, functions as a witness of the court rather than for either party, provides unbiased professional testimony, and thus encourages a fair judgment.

Summary

Problems peculiar to cardiology in industry are discussed.

The diverse problems of the various workmen's compensation law are presented briefly.

The classification or heart disease as related to industrial health is listed.

It is recommended that unity of opinion among cardiologists as to cardiac problems in industry be encouraged by the setting up of more expert panel plans and by continued action of special study committees.

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Glycolonitrile Toxicity

J. H. Wolfsie, M.D., Linden, N. J.

THE ORGANIC cyanides, or nitriles, as studied in animals in the toxicological laboratory, are a group of compounds of varying degree of hazard. Because of all combinations of a wide range of acute oral LD_{50}'s, of varying degrees of absorption through the intact skin (and even the conjunctiva), of varying degrees of local irritant effect, and of marked differences in vapor pressure, it is difficult to generalize as to the degree of hazard of these compounds as a group.

In animal studies with some of these compounds, e.g., acrylonitrile, cyanide ion was found in the blood. In other studies with a number of nitriles, the release of cyanide ion into the body fluids was inferred by the demonstration of serum thiocyanate values in proportion to the amounts of nitrile administered. Because of these experimental findings, of observations of the behavior of poisoned animals, and of observations of cases of illness from accidental human exposures in industry, the hazardous nitriles are believed to be toxic by virtue of the amount and rate of release of cyanide ion in the body. One interesting exception is B,B′-iminodipropionitrile, which was found capable of producing severe, permanent damage to the brain, eye, and genitourinary tract in experimental animals.

This paper is concerned with a discussion of the toxicity of glycolonitrile, an example of a highly toxic organic cyanide, because of the availability of both animal and human exposure data from which some interesting and useful observations can be drawn at this time.

Physical Properties

Anhydrous glycolonitrile, $HOCH_2CN$, has the structure formula $HO-\underset{\displaystyle H}{\overset{\displaystyle H}{\overset{|}{\underset{|}{C}}}}-C\equiv N$. It is a colorless, odorless liquid which is very soluble in water, ethanol, and ether. Its boiling point is 183°C at 759 mm/Hg. It is available commercially as a 70% aqueous solution stabilized at pH 2 to 3 to prevent polymerization. The partial pressure of glycolonitrile in such a solution is 0.015 mm/Hg at 30°C. Its vapor density is exceedingly small.

Animal Toxicity Studies

The following results are given on the basis of the anhydrous material, unless otherwise specified, although the studies were performed with 0.05% weight/weight aqueous solution.[1]

The acute oral (single feeding) LD_{50} for male albino mice is 10 mg/kg. All deaths occurred within two hours after dosing, and survivors made an uneventful recovery. The signs of intoxication observed resembled those of cyanide poisoning. The acute skin LD_{50} for albino rabbits lies between 105 mg/kg and 130 mg/kg. Mild dermal irritation of short duration was found in some of the survivors.

A single instillation of 0.05 ml of a 50% solution (26 mg) placed beneath the eyelid of each of three albino rabbits produced an immediate irritative effect of moderate degree. The animals exhibited evidence of systemic poisoning, including convulsions and coma, 15 to 30 minutes after dosing, and all were dead within 68 minutes of the start of the test.

Seven mice, seven rats, and seven guinea pigs were exposed to an average vapor concentration of 27 ppm of glycolonitrile in air for eight hours, at the end of which time mortalities were six of seven mice, two of seven rats, and none of seven guinea pigs. Signs of toxicity were minimal and consisted of lethargy and slight lacrimation. The remaining mouse and four of the rats died within 18 hours of termination of the exposure.

In a subacute feeding experiment with albino rats for 13 weeks, in which, at the highest feeding level, the males were ingesting 62 mg/kg/day and the females 92 mg/kg/day, no ill effects were observed. The sera showed amounts of thiocyanate related to the dose of

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glycolonitrile and generally showed no cyanide. Intraperitoneal injection of glycolonitrile into rats was followed by the prompt appearance in the blood serum of cyanide with a concomitant prompt increase in thiocyanate levels. The levels were proportional to the doses employed.

These data suggest that glycolonitrile is a highly toxic compound whose mechanism of action is related to cyanide liberation within the animal body with rapid detoxification involving conversion of cyanide to thiocyanate.

Case Reports

Two workers exposed to glycolonitrile by skin contact developed illnesses that were very much alike. Although there was no opportunity for detailed clinical study of these cases, such information as could be developed is presented for its possible usefulness to other physicians called upon to treat similarly exposed persons.

Case 1—A 30-year-old operator in a chemical manufacturing plant was seen at the hospital seven hours after he had reported to the plant dispensary.

By 6:35 p.m. on Sept. 25, 1959, he had finished filling 55-gallon drums with 70% aqueous glycolonitrile and had gone outside the building for a smoke. He did not notice any odors, and he was not aware of having come into contact with the substance or of any unusual circumstance. This glycolonitrile was filled through a stainless-steel corrugated filling line into drums at a drumming station which was adequately equipped with local exhaust ventilation. Because of the temporary nature of the filling line, operators were holding it under their arms and close to their sides when making final weight adjustments and capping the drums. A leak developed in the filling line at its connection, resulting in the glycolonitrile spiraling its whole length. As the operator held this line close to his body, the glycolonitrile contaminated his clothing and thus allowed for skin absorption.

About 5 to 10 minutes after he had finished the operation, he suddenly experienced a headache and then dizziness, unsteady gait, and rubbery legs, followed by vomiting. He did not believe that he had been unconscious or had fallen, but he remembered walking to the plant dispensary, taking a shower, and receiving an injection from the physician. His memory otherwise was a blank.

On admission at 7:40 p.m., he was rather pale, somewhat bewildered, and perspiring moderately. He was put to rest in bed and given supportive treatment. His pulse rate was 104 per minute, and the pulse was of good quality. Respirations were 24 per minute. He vomited several times.

Although the patient stated he did not get any material on his skin or clothing, his foreman, after careful examination of the patient's coveralls, believed that he detected glycolonitrile on the side of the garment. At 8:45 p.m., he was made to inhale, intermittently, the contents of one ampule of amyl nitrite, and he was given a thorough soap-and-water shower bath.

By 9:15 p.m., vomiting had stopped and the nausea had subsided but the patient spoke irrationally and became increasingly unresponsive. He was given 100% oxygen by face mask and he became alert, but he soon became unresponsive again, with the radial pulse irregular and rapid.

At about 9:45 p.m., after the intravenous administration of 30 cc of 25% sodium thiosulfate, he responded well and remained clear from then on. Subsequent to this latter medication, he developed a severe chill, which lasted 15 minutes.

When seen by the author at 1:10 a.m. on Sept. 26, he was resting comfortably in bed and appeared well. He was clear, bright, rational, and well oriented. The findings on physical examination were not remarkable.

At 8:30 a.m., the oral temperature was 99°F, the pulse rate 84, and the respiration rate 20 per minute. When seen again by the author, at 9 a.m., he stated that he felt well and he enjoyed his breakfast. On examination, there were no significant findings, and he was discharged from the hospital.

When he returned to work at 11 p.m. on Sept. 26 he reported to the nurse that he had rested in bed at home all day, abstained from lunch, but vomited after eating a light dinner earlier that evening. At the time of reporting, he complained of weakness and nausea. His face was flushed; the temperature was 98.6°F, pulse 88, and respirations 24. He wished to return to work, and he was assigned to a very light job. His symptoms persisted through the night.

When he reported to work on his next shift, at 11 p.m., Sept. 27, he stated that he did not feel well. He had eaten a light breakfast that day but was unable to eat any other meal because of nausea. He also complained of general weakness.

At 8:45 a.m. on Sept. 28 he continued to complain of some weakness and nausea, but stated, "otherwise, I feel fine." On physical examination by the author, he appeared well and the only abnormal finding was a mildly injected pharyngeal mucosa. He was seen finally on the morning of Oct. 1, at which time he no longer had any complaints. On examination, he appeared recovered and he was discharged.

In the second case the illness was brought to our attention after recovery had taken place.

Case 2—On Sept. 24, 1959, a 36-year-old operator in a chemical manufacturing plant filled 55-gallon drums with 70% aqueous glycolonitrile from about 6 until 9 p.m. He was using the same filling line that the operator in Case 1 used on the following evening, and the drums were being filled under adequate local exhaust ventilation. He was holding the filling line under his left arm and against his coveralls, and he was aware that 12″ × 6″ area on the left side of his coveralls was getting wet with the product while performing this job. There was no stinging or burning sensation of the skin; it just felt a little cool and wet. By the time he

removed his clothing prior to taking a soap-and-water shower bath (10:30 p.m.), he noticed that this area of the coveralls was almost dry. He left the plant at 11:05 p.m. feeling well and walked to the parking lot, about one block away.

When he reached his car, he began to feel weak and dizzy. He had experienced similar weakness on previous occasions, unrelated to any chemical exposure, from which he obtained relief by drinking a bottle of a cola drink and so he did not consider this episode as different from the others. When he sat in his car, he felt well, and he drove without trouble for about three miles to a gasoline station on the highway. When he got out of his car, he was dizzy and weak and his gait was unsteady, "as if you don't know where you're going." After drinking a bottle of a cola drink, he became nauseated and returned to his car. He drove home (approximately a mile and a half) with a great deal of difficulty: "I don't know how I got home, because I don't remember going through lights or anything. The next day, I found my car parked with two wheels on the sidewalk."

After arriving home, he vomited and then lay down for about a half-hour and had the sensation of the room spinning around. He had no headache. He arose again because of severe nausea and retching. He went back to bed and applied extra blankets because he felt chilly and apparently fell asleep until 10 o'clock the next morning, when he awoke soaking wet and feeling weak and "washed out," but he no longer had vertigo or nausea. He had no appetite and did not eat that day. He reported to work at 3 p.m. on Sept. 25. The weakness lasted until after he ate, which was about 4:30 p.m., and after that time he had no further symptoms.

Comment

These cases were most probably the result of skin exposures to glycolonitrile. Although hydrogen cyanide is used in the manufacture of the nitrile, the glycolonitrile had been made on a prior shift and, when tested, showed no remaining free hydrogen cyanide. The product was drummed under local exhaust ventilation, which was checked and found adequate. The manner of skin contact was later determined when the leak in the filling line was discovered. None of the material spilled onto the floor but, instead, soaked into the men's clothing. The vapor pressure of glycolonitrile in a 70% aqueous solution is exceedingly small; hence, in the event of a spill, it would not lead to significant atmospheric concentrations.

Except for the delay in onset of symptoms, these cases resemble the illness which follows skin absorption of hydrogen cyanide: a greater duration of the acute stage, relatively less response to antidotal therapy during this stage, and a subsequent period, which may last a number of days, during which the patient does not feel well. By contrast, the patient who is acutely ill as a result of hydrogen cyanide by inhalation responds well to antidotal therapy and has no persistence of symptoms.

Summary

Glycolonitrile has been shown by animal experiments to be a highly toxic compound whose mechanism of action is related to liberation of cyanide, which, in turn, is converted to thiocyanate. Because of its physical properties, glycolonitrile is expected to present a hazard by skin absorption rather than by inhalation. Two workers (one of whom was treated) who had substantial skin exposure experienced illness characterized by delayed onset, failure to respond as expected to antidotal therapy (in the treated case), and persistence of symptoms in gradually diminishing degree with the passage of time.

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Industrial Methemoglobinemia

H. C. Halsted, M.D., New York, N.Y.

THE PROBLEM of methemoglobinemia remains one of contemporary interest and significance. There are three principal reasons for this: the widespread industrial use of the nitro and amino derivatives of the aromatic series; the high degree of hazard many of these compounds have for humans; and the fact that there are still many aspects of the problem in which scientifically established fact has not yet displaced numerous empiricisms and assumptions. At the outset, I should like to establish one reference point necessary to the identification of the problem at hand. While there are numerous other potential causes of methemoglobinemia (inorganic nitrites, aliphatic nitro compounds, quinones, etc.), I will confine this discussion to that caused by the aromatic nitro and amino compounds. This restriction is not at all an arbitrary one, as almost without exception the cases of methemoglobinemia, or "blue lip," seen in industry are due to overexposure to one of the two latter groups of compounds. Chemically this series is characterized, of course, by the substitution of the amino (NH_2) radical or of a nitro (NO_2) radical for a hydrogen atom in the benzene ring or in one of its homologs.

Aromatic nitro and amino compounds are used extensively as intermediates in the synthesis of "aniline dyes," in pharmaceutical manufacture, and as accelerators and antioxidants in the rubber industry. They find additional use in the production of paints and varnishes, plastics, and fungicides, and in many other areas.

While it is manifestly impossible to attribute a single constant and universal toxic property to such a large variety of compounds, nevertheless the ability to cause methemoglobinemia to one degree or another characterizes a sizable number of compounds of this group.

Chemistry and Physiology

A brief consideration of some of the biochemical mechanisms involved is essential to a better understanding of the nature of methemoglobinemia. Hemoglobin, the normal blood pigment, is the chief coloring matter of erythrocytes. It is a conjugated protein consisting of a colorless protein of the histone class, globin, and an iron-containing pigment portion, ferroprotoporphyrin, or heme. The pigment, or heme, portion of this complex bears further scrutiny, for it is here that the biologically significant reactions we will shortly discuss occur. Heme is itself a complex consisting of iron and protoporphyrin, a so-called Type III porphyrin. The porphyrins are pigments which are found throughout plant and animal life, from the highest to the lowest forms. One porphyrin is the pigment basis of chlorophyll, while another is found as a brown pigment in the shells of many eggs. Without attempting to enter into a lengthy discussion of porphyrin chemistry, it should be noted that the biosynthesis of porphyrin includes participation of the amino acid glycine via the intermediates δ-aminolevulinic acid and porphobilinogen. The steps involved in the subsequent conversion of porphobilinogen to protoporphyrin are not yet completely understood.[17]

The ability to combine loosely with oxygen so that it is readily released is a peculiar and essential property of hemoglobin. Sollman[13] states that "hemoglobin is unique among iron compounds in that it can combine reversibly with oxygen, without oxidation. The iron of hemoglobin reacts reversibly with molecular oxygen, one molecule of oxygen for each atom of iron, to form oxyhemoglobin." This process, called oxygenation, is not a true oxidation. No change of valence has taken place. The iron remains in the ferrous (Fe^{++}) state throughout. It is the globin which endows the iron-pigment part of hemoglobin with the unique property of forming a loose combination with oxygen. This property is lost if denaturization of the protein occurs. When the oxygen pressure in contact with hemoglobin is sufficiently raised, then oxygen is taken up—but no true oxide is formed.[1]

Methemoglobin, on the other hand, is an oxi-

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dized form of hemoglobin. One atom of oxygen combines with one atom of iron in a true oxidation reaction, with bivalent ferrous iron being converted to the trivalent ferric state. Significant here is the fact that oxygen is now tightly bound and not available for tissue use. Important also is the fact that, while the reaction is reversible, the enzyme systems within the erythrocytes only slowly cause the reduction of methemoglobin back to hemoglobin. This is usually not complete, however, as small amounts of methemoglobin are normally present in the blood at levels of 1% to 2%.

The tissue hypoxia which exists in the presence of an elevated methemoglobin is due not alone to the presence of this abnormal blood pigment, with the consequent loss of oxygen-carrying capacity of the blood. Coexisting with this is the fact that the residual oxyhemoglobin also becomes less capable of dissociating or unloading its oxygen to the tissues. This unfavorable shift of the oxygen dissociation curve is noted with other toxicants—carbon monoxide, for example. In the latter instance, however, carboxyhemoglobin exhibits a far greater capacity to bring about this unfavorable shift. It has been estimated that approximately twice as high a concentration of methemoglobin as carboxyhemoglobin is necessary to bring about a comparable shift in the dissociation curve of the residual hemoglobin.

Without questioning the validity of the basic mechanisms discussed in the above paragraphs, there is, nevertheless, evidence that other hemoglobin derivatives are also concerned in the production of chemical cyanosis.

There is mounting evidence that the simple reaction oxyhemoglobin to methemoglobin fails to account for all of the changes that take place when hemoglobin undergoes oxidation. The presence of other intermediate hemoglobin complexes has been firmly established by numerous investigators. These complexes may well be, in fact, methemoglobin precursors, and their existence raises some interesting speculations.

The ability of phenylhydroxylamine and nitrosobenzene to form methemoglobin is well known. Jackson and Thompson[10] utilized this property in a unique bit of in vitro research when they tagged the iodo derivatives of these two compounds with radioactive iodine (I^{131}). These materials combined firmly with hemoglobin, and so the erythrocytes became effectively labeled when the radioactive iodo compounds were used. They were able to identify by this method numerous methemoglobin precursors. Not only did they establish their existence, but they further demonstrated that these precursors were present without any apparent change in the absorption spectrum of the pigment. Thus, by conventional spectrophotometric analysis, these hemoglobin complexes would remain undetected.

Jackson and Thompson concluded that reactions like this "may be of importance in relation to the known toxic effects of aromatic nitro and amino compounds used in industry and medicine, for such substances may be metabolized to hydroxylamino and nitroso compounds, which could be responsible for the observed toxic effects."

Evans, Charsha, and Linch[4] elaborated further on this thesis and found support and explanation for some of their own observations. They had become convinced that a latent cyanosis existed in certain persons who were exposed to the aromatic nitro-amino compounds which defied detection by spectrophotometric analysis. The existence of certain hemoglobin derivatives which did not alter the absorption spectrum supported these convictions.

These are interesting observations and might well serve to implement and refine the preventive medical approach to poisoning by the aryl nitro and amino compounds. For the present, however, until closer correlation with actual clinical management of exposed personnel can be made, we must continue to rely on the conventional spectrophotometric method for guidance in the medical care of these persons. This method, it should be noted, is quite satisfactory to permit intelligent management of chemically cyanosed personnel.

Mode of Action

In considering the mode of entry, biological effect, and subsequent elimination of these aromatic nitro and amino compounds, we will discuss briefly one compound, aniline, as the prototype.

Aniline, or aminobenzene, is a nearly colorless oil with a specific gravity of 1.022. It solidifies at –6.2°C and boils at 184.4°C. It is slightly soluble in water but freely soluble in alcohol, ether, and other organic solvents.

Being fat-soluble, aniline (or its derivatives) readily penetrates the intact skin. It does so with almost remarkable ease and rapidity. A small amount spilled on the skin or clothing and not immediately and completely removed will soon give rise to signs of intoxication. While a categoric assertion here is hard to substantiate, it appears that skin absorption is the important mode of entry of the material into the body.

Following as a close second is the respiratory route. Aniline vapors are readily absorbed

through the vast absorptive surfaces of the lung, and just as serious overexposure can result from vapor inhalation as from skin contact.

Ingestion is of negligible importance as an industrial hazard.

Once the toxicant has gained entry into the blood stream, its exact action is somewhat obscure. Certainly we know that methemoglobin is formed, though the precise mechanism remains in doubt. It has been demonstrated that a sizable portion of these aromatic nitro and amino compounds do not form methemoglobin in vitro—or do so very poorly. Yet these same compounds in vivo are essentially active formers of methemoglobin.[2] The likelihood exists, then, that active intermediate metabolites are formed which are the real oxidants of hemoglobin.

Evans, and his associates,[4] commenting on the same point, state that "most of the aromatic amino and nitro compounds probably are not in themselves cyanosis producers, but biochemical redox processes create derivatives which hinder oxygen transport to the tissues by forming hemoglobin complexes."

Hamblin[7] discussed the role of these metabolites and concluded that "whatever the mechanism, metabolically, certain compounds are methemoglobin formers par excellence, such as p-dinitrobenzene, phenylhydroxylamine, p-aminopropriophenone and nitrosobenzene. Their marked effectiveness has been attributed to their intermediary metabolites, without very detailed substantiating evidence.

Recognition, Treatment and Control

Poisoning by one of these cyanosis-producing compounds is still encountered frequently in spite of vigorous control measures. Fortunately, the great majority of cases seen in industry are mild and transient and require little or no active medical treatment. It is usually an alert supervisor or co-worker who first notices the characteristic sign of blueness of the lips in an overexposed individual. Indeed, a careful indoctrination of both supervisor and worker is an important phase of any comprehensive control program. This training should be directed at both an explanation of the nature and potential danger of methemoglobinemia and what to look for to detect early cases.

If industrial hygiene precautions are not adequate or potential exposure remains high in spite of all possible precautions, it is wise to resort to routine checking of all exposed personnel. This can be effected by requiring each individual to report to the medical department at the completion of his work day. A nurse can then rapidly check lips, tongue, nail beds, etc., for early signs of cyanosis. If none has developed in the period devoted to showering and changing into street clothes, the chances are that no serious poisoning will develop later.

Mild Intoxication—The mild cases (methemoglobin 15%-25% or below) often go undetected. It is extremely difficult to note the characteristic sign of cyanosis of the lips, tongue, nail beds, etc., with a reading of 15% or less. A person with such intoxication rarely exhibits subjective symptoms; he sometimes exhibits a mild euphoria and seems puzzled and hesitant when notified that medical attention is indicated. Not infrequently, a bit of "selling" is necessary to convince him that he is indeed a patient and in need of medical attention.

Once the condition is recognized, a series of separate but interrelated actions should be set in motion. The prime requisite here is speed. A sample of blood is obtained for methemoglobin determination, which serves to establish both the initial severity of the exposure and a reference point to guide future management of the patient. It is normally repeated at intervals of approximately 60 minutes until a declining methemoglobin level is well established. An exposure history should be obtained as soon as possible. This should include the type of material handled, route of exposure, length of exposure, and whether or not fresh clothing has been obtained and a shower taken. It is also wise to obtain from the person the names of his co-workers (who may have had a similar exposure) so that they can be called in and checked. This will often obviate a serious poisoning by getting to a person before severer intoxication is manifest.

Treatment at this level (15%-25% methemoglobin) is directed primarily toward preventing further absorption of the toxicant. There are no halfway measures acceptable here. The person, whether or not he has had a previous shower, must be thoroughly showered again with soap and warm water. This should include a hair shampoo and a cleansing of the ear canals and nostrils, so far as is possible. Particular attention must be paid to the fingernails. The use of an orangestick and nailbrush will help to remove thoroughly any material collected there.

After this, a complete change of clothing must be obtained. It is not unusual for additional absorption to occur from the continued wearing of contaminated work clothing. Caps and shoes remain the chief offenders here, as they are frequently overlooked.

After this has been accomplished, the patient is kept under observation, usually at bed rest,

and given sweetened beverages to drink. He is watched long enough, usually one to two hours, to establish the pattern of the methemoglobin curve. Once it has been determined that the curve is falling, he is permitted to leave the medical department with two restrictions as to his activity. If he is returning to work, it must be in an area completely free from additional exposure to any cyanosis-producing material. If, on the other hand, he is discharged to his home, he must be counseled to abstain completely from any alcoholic beverage.

The rationale surrounding the insistence that alcohol in any form be avoided remains somewhat obscure. It has variously been assumed that alcohol favored methemoglobin formation by some stimulant action on the oxidation mechanism. Or again, that it might possibly have some effect on the reduction of methemoglobin to hemoglobin.

Hamblin[7] commented that, because of the fat-solvent characteristics of many of these compounds, it is possible that they are partially stored in the body by lipoid deposits. Alcohol, then, might serve to liberate this stored material. Whatever the exact biochemical nature of this phenomenon, it has certainly been noted clinically that alcohol ingestion reawakens symptomatology and reverses the normal decline of methemoglobinemia. It should, therefore, be rigorously avoided.

It has been our practice to keep these persons entirely away from possible further contact with the aromatic nitro and amino compounds until the methemoglobin level has completely returned to normal.

Moderate Intoxication—Persons with levels of 25%-50% methemoglobinemia are classified as having a moderate degree of cyanosis. Often, these patients will complain of a headache—occasionally of mild vertigo and of some weakness. The pulse rate is moderately increased. It is rare for them to complain of other symptoms. Indeed, the disparity between the clinical appearance of the patient and his symptomatology can be puzzling to the uninitiated. In spite of the obviously cyanotic appearance of these persons, they rarely complain of or exhibit shortness of breath. Even at higher levels of intoxication (50%-60%), dyspnea is rarely noted while the patient is at rest. Exercise can, however, at these higher levels bring on some degree of exertional dyspnea.

In this moderately cyanosed group, oxygen is valuable adjunct therapy. Almost invariably the headache is relieved rather promptly. While oxygen does not seem to speed the conversion of methemoglobin to hemoglobin, its use is, nevertheless, indicated for all persons with a methemoglobin level in excess of 25%-30%. It most probably acts to saturate the remaining hemoglobin, thus relieving tissue hypoxia.

The therapeutic use of intravenous dextrose for cyanosed patients is a highly controversial issue. Bodansky,[2] after evaluating the available evidence, concluded that there was little physiological justification for its use. Fleming and colleagues,[6] on the other hand, note its routine use in those cases with a methemoglobin level between 40% and 60%. Mangelsdorff, in comparing various methods of treatment, felt that "the rate of conversion from methemoglobin to oxyhemoglobin is slightly faster when intravenously administered dextrose is used to speed up the normal reversible process." I am inclined to this latter viewpoint and feel it is realistically stated. In order to keep a proper perspective, however, it must be pointed out that even the warmest advocates of dextrose therapy claim little more for it than some slight hastening of the reversion mechanism.

Ascorbic acid in large doses has been utilized rather extensively in therapy. Its value as a reducing agent plus its wide margin of safety makes it, in theory, an attractive drug. It is not, however, the ideal therapeutic agent in all cases of methemoglobinemia. Its value has been repeatedly demonstrated in the familial idiopathic variety, where the need of prolonged administration makes it well suited for this type of therapy. In chemical cyanoses, however, I would reserve its use primarily for the moderately severe poisonings. For those more acutely ill, whose methemoglobin level is 60% or above, it becomes inadequate therapy because of its lack of ability rapidly to hasten the reversion mechanism.

These patients are checked hourly for methemoglobin level. There are times when a continually rising level necessitates a second complete cleansing. This is particularly true if doubt exists as to the efficacy and completeness of the initial effort. It must be remembered, however, that in some instances a continuing rise is due not to additional absorption but to the slow rate of conversion of hemoglobin to methemoglobin. In any case, once a peak has been reached and the methemoglobinemia begins to fall, the pressure is off all concerned. Almost invariably, the fall continues unchecked, and in 24-48 hours the methemoglobin level has returned to normal.

Severe Intoxication—Persons who are severely intoxicated (in excess of 50% methemoglobin) present a rather alarming picture. They are invariably deeply cyanosed and appear ap-

prehensive and acutely ill. They will typically complain of severe headaches, definite weakness, and occasionally vertigo. The pulse rate is considerably increased. Exertion causes some degree of dyspnea, though at rest this is rarely noted. In the upper levels of 60%-70%, persons will often exhibit a marked lethargy, which can progress to convulsions and coma if the methemoglobin level continues to rise. These patients constitute medical emergencies, and every effort must be made to assure prompt and vigorous treatment. While there is a leveling-off or state of equilibrium which seems to occur in the region of 75% methemoglobin, this does not constitute a barrier which cannot be exceeded if the exposure is sufficiently severe. The lethal level is probably somewhat slightly above this—in the neighborhood of 80%-85%.

Treatment, which must be prompt, consists of the routine supportive measures already mentioned plus one major variation. When the level of methemoglobin reaches or exceeds 60%, methylene blue is the drug of choice. The methemoglobin level must be brought down as rapidly as possible, and the intravenous injection of a 1% aqueous solution of methylene blue seems to accomplish this more effectively than any other medication. A low dosage of 1 to 2 mg per kilogram of body weight is recommended.

Methylene blue cannot be used indiscriminately, for its use is not without hazard. It is itself a methemoglobin former, and, further, it is capable of causing hemolysis of red blood cells. Too-rapid injection of the drug may produce hives and a burning sensation of the lips. Nadler, Green, and Rosenbaum[12] detected evidence of depression of the ventricular musculature in EKG patterns. This they concluded after noting either reduction in the height or inversion of the T wave, frequently with lowering of the R wave.

Why, then, in the light of the seeming paradox, is methylene blue advocated? The reason is simply this: Many of the untoward effects are the result of much higher dosages than those recommended for therapy. Bodansky notes that, while methylene blue is a methemoglobin former it does so poorly in vivo. Further, apparently the reduction reaction of methemoglobin to hemoglobin is far more active than is the opposing oxidation reaction. Wendel[16] comments on this by stating "the ability of methylene blue to accelerate the reduction of methemoglobin *in vivo* has been well established."

Numerous other authorities agree with this viewpoint and, while recognizing its hazards, nevertheless feel that methylene blue is the most effective medication available. Sroka[14] perhaps sums it up best by stating, "Even though use of methylene blue should be questioned because of its toxic nature, it is the most serviceable drug for methemoglobin detoxification." Provided it is used cautiously, in low dosages, and only in cases in excess of 60% methemoglobin, I concur completely with this conclusion.

Control Methods

We have seen that not only are these aromatic nitro and amino derivatives toxic to humans, but many of them, because of the facility with which they gain entrance to the body, are also highly hazardous. With such potential, constant and adequate control measures are indispensable. Without good industrial-hygiene precautions, the widespread use of these materials would be fraught with serious dangers to exposed personnel.

Wherever possible when used in the liquid form, closed systems are indicated. Here the worker exposure is nil, except for breakdown or routine maintenance work, where again a hazard exists to those working on the tools. This point should be borne in mind, for we have occasionally found serious overexposures resulting from repair work done on a presumedly safe "closed" system.

Where inhalation of vapors is possible, again careful control measures must be provided for. Adequate ventilation is essential at all times, as the vast absorptive area of the lung gives ready entrance to atmospheric contaminants of this sort.

Wherever skin contact is possible, adequate protective clothing must be worn. The use of gloves, rubber boots, and daily laundered clothing is essential. We have used neoprene-dipped cotton gloves and vinyl-plastic-dipped canvas gloves and feel that they both offer reasonably adequate protection. It must be noted, however, that ordinary rubber fails to give complete protection against the aromatic nitro and amino compounds. Fleming and others[6] point out in their textbook that really only synthetic butyl rubber seems completely satisfactory for complete protection against these compounds. Difficulty in fabricating into gloves, however, has been a deterrent to its widespread adoption. An interesting innovation along these lines has been the development of a rubberized suit which completely covers the worker, head to toe. It has a fresh air supply which is pumped in from an uncontaminated source near the work area. Unfortunately, it has one serious

drawback. The performance of manual labor for any extended period of time is difficult because of a rapid fatigue which develops in the suited worker. Its use thus seems limited to an occasional troubleshooting repair job of short duration. As a corollary to this, we have found that control measures seem most apt to break down during the hot, humid days of the summer. We expect and seem to get our worst cases during hot weather. This is perhaps quite logical if one considers the increased vapor pressure in both the liquid and the solid members of the group and the inevitable omission of proper protective clothing on hot days. Whether these alone account for the increased incidence of methemoglobinemia or other factors enter into it, the fact remains that a tightening of control measures is indicated during hot, humid weather.

Summary

Methemoglobinemia, the primary effect of poisoning by the aromatic nitro and amino compounds, has been considered.

A review of the basic biochemical mechanisms involved in the production of methemoglobin from the normal blood pigment, hemoglobin, was discussed.

Essentially from the standpoint of personal observations we have further considered the varying aspects of recognition, treatment, and control of chemical cyanosis.

Watchful waiting plus general supportive measures are adequate for the treatment of mildly or moderately cyanosed persons.

Patients who are severely cyanosed require vigorous and adequate medical therapy. Methylene blue is recommended as the most valuable single drug available to bring about the rapid reduction of methemoglobin to normal hemoglobin.

Careful attention must be given to adequate industrial hygiene control measures for whenever the aromatic nitro-amino compounds are handled they constitute a constant hazard to exposed personnel.

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The Injured Knee

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TREMENDOUS advantage in the accurate diagnosis of the injured knee is held by the first examiner, who sees the patient minutes or a few hours after the accident. Usually, it is the industrial physician, the team doctor, or the family physician who enjoys this tactical advantage, and his ability adequately to assess the injured knee is frequently the prime factor in deciding the outcome.

The early examiner can usually obtain the best history of injury, either from the victim or from witnesses. Points of tenderness are clearly defined. Instability due to serious ligamentous damage is not masked by muscle spasm. The picture has not been confused by well-meaning but misdirected efforts at treatment.

Truly, these are the golden hours for determining the exact nature and extent of damage to the joint, and upon this vital evidence rests effective treatment.

Functional Anatomy

Ligaments—The knee, for practical purposes, may be considered a hinge joint which allows some rotatory motion of femur on tibia in all positions except full extension.

Lateral wobble in the hinge is prevented by medial and lateral collateral ligaments. The lateral ligament, extending from the lateral epicondyle of the femur to the head of the fibula, is rarely injured. The medial collateral ligament, on the other hand, is involved in virtually every sprain of the knee and figures in the differential diagnosis of more than half of all knee injuries.

Roughly triangular in shape and double-layered in construction, the medial collateral ligament anchors by its apex to the medial femoral epicondyle about an inch above the joint line and by its base to the tibia about an inch below the joint line. It is designed to pivot on its apex as the knee flexes and extends to maintain a modicum of lateral stability in all positions of the joint. Its glide path over the deeper fibers, which attach to the joint capsule and the medial meniscus, is friction-proofed by a bursa, which may become a source of knee symptoms.

As a rule, ligaments tear at their point of insertion into bone. Maximum tenderness in medial collateral ligament sprains is therefore likely to be about an inch above or below the joint line if the knee is examined soon after the accident. As time passes, however, the tenderness diffuses, and this valuable differential diagnostic point is lost.

Severe or continuing spraining force may break through the first layer of the ligament and tear the deep fibers, in which case joint-line tenderness, effusion into the joint, and combined injuries to the medial meniscus and to other structures may be present.

Fore and aft sliding of the tibia on the femur at the knee hinge is prevented by a pair of short ligaments, the cruciates, which crisscross the space between the femoral condyles and the tibial spines. Taking their designation from their tibial insertions, the anterior cruciate crosses from the anterior tibial spine backward and laterally to the back of the lateral femoral condyle and keeps the tibia from shifting forward on the femur. The posterior cruciate ligament forms the opposite leg of the letter X and prevents the tibia from sliding backward on the femur.

Isolated injury to the cruciate ligaments is uncommon. The clip, in football, is an example of an injury mechanism which may rupture a cruciate ligament. Usually, however, cruciate ligament damage occurs as an extension or prolongation of an injuring force which disrupts the medial collateral ligament.

The medial meniscus and the lateral meniscus are half-washers, wedge-shaped in cross section, applied to the margins of the respective tibial

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plateaus converting these flat areas into shallow cups to receive the convex femoral condyles. Avascular except for a narrow peripheral zone of attachment, the menisci have extremely low healing potential.

Menisci are torn by torsional stress on the partially flexed weight-bearing knee. Once torn, they almost never heal. A diagnosis of torn meniscus is, with only slight exaggeration, a ticket to the operating room or a pass to permanent partial disability—sometimes, both.

Quadriceps—The quadriceps-patella mechanism, in addition to its obvious function as the prime extender of the knee, plays a vital role in stabilizing the joint. Its tendinous expansions fan out to cover the front 180 degrees of the knee joint capsule. A strong, tensed quadriceps can effectively stabilize the knee, even in the absence of collateral and cruciate liagments. Loss of quadriceps tone and power can result in symptom-producing instability of the knee, even in the presence of intact collateral and cruciate ligaments.

Among features common to all knee injuries, rapid wasting of the quadriceps muscle is of outstanding importance. Probably as the result of pain, a reflex inhibition of the muscle occurs, and atrophy develops with startling rapidity. Unless actively combated by enforced quadriceps exercises, the weakening and wasting of the muscle can, in itself, produce a vicious type of self-perpetuating knee disability.

The quadriceps insufficiency allows ordinary twists and stresses to penetrate the protective muscular barrier and produce repeated stretchings of ligaments and capsule and even occasional nipping of the synovial membrane. This results in pain and effusion which, in turn, produces further reflex inhibition of the quadriceps and completes the cycle.

Exercises for the quadriceps muscle must be started at the earliest possible moment following the knee injury. Straight leg raising, which involves no motion of the injured joint, can usually be instituted on the day of injury. It may take considerable time to train the patient to perform even this simple action, so powerful are the inhibitory forces. Whatever time, whatever tricks, whatever repetition is necessary must be used to institute active contraction of the quadriceps for a least five minutes of every waking hour from the outset of the disability. Physical therapy is no substitute for active exercise, and no one can teach the exercises as effectively as the doctor in charge of the case.

As healing of the knee injury progresses, knee-motion exercises can be added, and, still later, heavy resistance knee-extension work begun. Quadriceps exercises must become a constantly repeated part of the patient's existence, however, until midthigh circumference on the two sides is equal.

If there is a single key to the effective management of the injured knee, it is conservation and restoration of full quadriceps efficiency.

Effusion

Effusion into the joint is another feature common to all knee injuries above a certain degree of severity. As a matter of fact, presence or absence of effusion can be used as a rough index of the severity of injury. If no effusion occurs, the injury can be presumed to be relatively slight.

Since effusion is a constant feature of most serious knee injuries, traumatic synovitis must be a diagnosis of exclusion, made only after painstaking history and examination have eliminated the possibility of damage to the joint supportive structures. It is as dangerous to make a diagnosis of traumatic synovitis of the knee without thorough inventory of the ligaments and menisci as it is to diagnose sprain of the wrist without an x-ray film of the carpal scaphoid.

The knee-joint capsule, in addition to sheathing the articular surfaces, extends for a hand's breadth above the patella as the suprapatellar pouch. When there is increased fluid in the joint, the pouch is clearly visible, and it is the presence of fluid in this extension of the joint cavity up under the kneecap which allows ballottement of the patella, a time-honored physical sign of effusion. The suprapatella pouch provides a handy backwash where the joint may be aspirated without fear of injuring the articular surfaces. Often erroneously considered a bursa, the pouch is an anatomically constant extension of the true knee-joint space.

Bursa Involvement

A formidable collection of bursae do inhabit the knee region, and a number of them may be involved in traumatic situations. The prepatellar bursa, of housemaid's knee, lies between the patella and the skin. Below the joint line anteriorly are two other bursae which, like the prepatellar bursa, may become swollen and inflamed in workers who kneel. The pretibial bursa lies deep between the patellar tendon and the tibia, and the tibial tuberosity bursa lies between that bony prominence and the skin.

The medial collateral ligament bursa, already mentioned, lies beneath the leading edge of the ligament close to the joint line and may become

inflamed and irritated after activity in a position of full knee flexion. A bursa posterior in relation to the semimembranosus muscle of the medial hamstring group, when distended, is known as Baker's cyst or popliteal cyst. Irritation of this bursa may follow long periods of work with the knee forced into full extension, as in working on a ladder.

History

Adequate evaluation of the injured knee depends first upon an accurate and detailed history. The amount and direction of the force must be judged, and the position of the leg when the force was applied must be known.

Frequently, the severity of injury can be gauged by what the patient was able to do immediately afterwards. Was he able to get to his feet without help? Did he, indeed, fall to the ground at all? Did he continue at work or in the ball game? If so, how long before the knee became disabling?

Time of appearance of effusion is valuable evidence. Massive distension of the capsule within an hour following the injury means tearing of synovial blood vessels and hemarthrosis. The increased production of synovial fluid associated with traumatic synovitis ordinarily does not produce detectable swelling for at least six to eight hours.

A history of locking should be sought but interpreted circumspectly. True locking means meniscus tear, if intar-articular loose body can be excluded. True locking implies a bony-firm block to extension of the knee, oftentimes suddenly and audibly relieved by some special maneuver of the leg usually involving further flexion of the knee, rotation of the tibia on the femur, and then sudden extension of the knee.

The patient, however, may describe any difficulty with extension of the knee as locking. A tight effusion will make extension of the knee difficult, and the patient may announce that his knee is locked. Similarly, hamstring spasm may be reported as locking. Even the subpatellar crepitus and discomfort of chondromalacia patellae may be referred to as locking by the patient.

The position of maximum comfort of the knee is occasionally important. Most freshly injured knees are most comfortable in 20 to 30 degrees of flexion. This position relieves the pull on a sprained medial collateral ligament, eases the tension of a tight effusion, and provides maximum relaxation for spastic hamstrings. If full extension is the position selected for comfort, an injury to the quadriceps-patella mechanism is suggested.

Finally, in the history, it is helpful to know whether the patient has had previous difficulty with the injured knee. Old meniscal tears are particularly likely to produce recurrent periods of acute disability following twisting injuries. Especially in the older age group, a history of arthritis or gout should be sought, particulary if there is a significant lag phase between the traumatic incident and the appearance of disability.

Examination

Examination of the injured knee should follow a definite routine and should be made with full exposure of both legs and with frequent reference to the uninjured knee. Since lateral and anteroposterior stability and even range of motion vary so widely from one person to the next, the uninjured knee is the only normal standard of comparison for any patient.

Inspection of the knee may reveal external evidence of violence which will amplify or modify the notion of injury obtained from the history. The presence or absence of effusion and of soft-tissue swelling should be noted, and one should be distinguished from the other. A minor tear of the medial collateral ligament may produce considerable soft-tissue swelling along the medial side of the joint without arousing particular concern in the mind of the examiner. If, on the other hand, the history of injury indicates damage to the medial supporting structures and there is effusion in the joint, the yellow flag goes up. Damage has almost certainly occurred to the deep fibers of the ligament, there may be meniscus damage in addition, lateral stability may be impaired, and if the force was sufficiently great or prolonged, the anterior cruciate ligament may be injured with loss of anteroposterior stability.

Massive effusions can be diagnosed by ballottement of the patella. Lesser amounts of fluid can be detected by compressing the suprapatellar pouch with the flat of one hand and using the thumb and forefinger of the other hand placed on either side of the patellar tendon on the joint line to transmit and receive a fluid wave.

The range of motion in the injured knee is checked and compared with the normal knee. Decision must be reached, if possible, as to the nature of any block to full extension. It is usually not too difficult to distinguish the squishy feel of an effusion block from the slowly yielding pull of spastic hamstrings or the medial pain of a stretched collateral ligament from the bony-hard block of an incarcerated meniscus.

Examination for lateral instability is best done first on the uninjured knee, both to establish the normal for the patient and to reassure him as to the nature of the next step in the examination. Testing the integrity of the medial and lateral collateral ligaments must be done with the quadriceps relaxed and with the knee flexed 15 or 20 degrees to relax the cruciate ligaments. The femur is firmly grasped with one hand, the tibia with the other, and an effort made to rock the tibia medially and laterally on the femur. Any lateral instability in excess of that present in the normal knee is noted in degrees. In the usual medial collateral ligament sprain, stressing the tibia laterally in relation to the femur will produce pain along the medial ligament as its fibers are stretched. Conversely, in the usual medial meniscus injury, lateral stress of the tibia on the femur may relieve discomfort as the medial joint space is widened, but pain may be produced as the tibia is stressed medially in relation to the femur, narrowing the medial joint line and pinching the injured meniscus.

Anteroposterior stability is best investigated with the patient in a sitting position, his legs hanging over a table edge. The tibia is firmly grasped with both hands and pushed backward and pulled forward on the fixed femur. Forward slide of tibia on femur, the so-called drawer sign, indicates damage to the anterior cruciate ligament. Backward slide of tibia on femur implicates the posterior cruciate. Again, comparison must be made with the normal, uninjured knee.

The point or points of maximum tenderness should be localized as accurately as possible. In general, fresh sprains will show tenderness about an inch above or below the joint line; fresh meniscus tears will usually have maximum tenderness on the joint line just medial to the patellar tendon.

Although seldom of importance in the freshly injured knee, the determination of thigh circumference at identical levels on the two legs is essential information in the evaluation of the patient with continuing complaints following a knee injury. It is probably safe to state that any significant knee disability present for more than a week will produce quadriceps atrophy and definite measurable shrinking of the involved thigh. Thigh circumference, then, becomes a handy objective ruler by which degree of disability may be evaluated in the patient with continuing complaints but with minimal or confusing findings in the knee.

The remaining standard examination maneuvers for the injured knee are chiefly concerned with producing detectable false motion of a torn meniscus are probably neither necessary nor advisable in the freshly traumatized joint.

Evaluation of the injured knee should always include x-ray examination. Plateau fractures of the tibia, avulsion of the tibial spines, and fractures of the patella are most commonly encountered. Occasionally, stress x-rays may be necessary to settle a question of lateral instability, but in most cases this should be determinable clinically.

Laboratory studies on blood and synovial fluid assume importance in the workup of the chronic knee but are not often critical in the fresh-injury situation.

Management of Industrial Knee Injuries

In a recent review of 1,000 consecutive compensation cases involving orthopedic conditions at Eastman Kodak Company, 72 knee injuries were encountered. The accompanying Table shows the diagnostic breakdown in these 72 cases.

Sprain was the most common of the knee injuries, and, of 27 sprains, 25 involved the medial collateral ligament. Sprains may vary in severity from a minor stretch to a complete avulsion, the presence of effusion ordinarily being the tipoff on the more severe injuries. Except in the extremely extensive sprains, tenderness is usually maximum over the upper attachment of the medial collateral ligament. Pain is increased by stressing the joint in the knock-knee position.

Treatment of the common medial collateral ligament sprain depends entirely upon severity as demonstrated by degree of lateral instability. As in all fresh knee injuries, cold compresses should be used for the first 48 hours, and quadriceps setting exercises should be started at the earliest possible moment. If there is no detect-

Diagnosis in 72 Consecutive Knee Injuries

Sprain		27
Medial collateral ligament	25	
Lateral collateral ligament	2	
Tear of meniscus		20
Medial meniscus	19	
Lateral meniscus	1	
Contusion with hemarthrosis		8
Prepatellar bursitis		6
Tenosynovitis, patellar tendon		3
Chondromalacia patellae		3
Traumatic synovitis		2
Fracture of patella		1
Baker's cyst		1
Medial collateral ligament bursitis		1
Total		72

able lateral instability, a simple adhesive strapping or elastic bandage for a period of about three weeks is sufficient support.

If there is lateral instability, fixation in a plaster cylinder for six to eight weeks is indicated. If there is suggestive evidence of complete avulsion of the ligament with effusion in the joint, market lateral instability, and perhaps some evidence of meniscus or cruciate ligament damage, the patient should have the benefit of expert consultation for consideration of surgical repair.

Tears of the menisci were next most frequently encountered, and, of the 20 tears included in the series, 19 involved the medial meniscus. Most of the medial meniscus tears resulted from the usual torsional stress plus abduction on the partially flexed weight-bearing knee. Several occurred, however, on attempt to straighten the knee after a prolonged period in the full-kneeling position. If a worker spends a considerable time kneeling and actually sitting on his heels with his body weight forcing his knees into full flexion, the medial meniscus tends to slide out of position and may be caught and split by the medial femoral condyle as the knee is extended.

Tears of the meniscus are almost always accompanied by effusions, and the point of maximum tenderness in the common medial meniscus tear is on the joint line just medial to the patellar tendon. There is frequently an extension block, and stressing the knee in the bowleg position often increases the pain.

Once a firm diagnosis can be reached, the treatment of a torn meniscus producing significant disability is surgical excision, preceded and followed by quadriceps exercises.

Eight of the knee injuries were simple contusions, usually to the front of the joint, followed by hemarthrosis. Ice packs and compression bandages are indicated in the first 48 hours, and tight effusions should be aspirated through the suprapatellar pouch. Quadriceps exercises should be encouraged.

Direct blows to the front of the knee or rolling on the flexed knee may stir up irritation in any of the three anterior bursae. In this series, six knees were seen with acute traumatic bursitis involving the prepatellar, the pretibial, and the tibial tuberosity bursae. Aspiration of the bursa, injection of the bursal sac with local corticosteroid, and temporary modification of activity have usually been successful. Failure of treatment or frequent recurrence may force excision of the bursa.

The same type of injury to the front of the knee occasionally produces a tenosynovitis of the patellar tendon marked by extreme pain, swelling, redness, and characteristic "wet leather" crepitus. Immobilization of the knee in full extension and administration of corticosteroids, locally or systemically, have been effective treatment.

Several knees injured by direct impact to the front of the joint developed no severe immediate disability but, after a lag phase varying from one to eight weeks, became painful and crepitant. A diagnosis of chondromalacia patellae was made. Following a direct blow over the patella, degeneration of the articular cartilage on the back of the kneecap may occur and the gliding surface become rough and pitted. Subpatellar pain and crepitus result, and the symptoms are aggravated by activities which place the quadriceps-patellar mechanism under tension and squeeze the kneecap back against the underlying femoral condyles. Stair-climbing and rampwalking become painful, although standing or walking on the level is seldom bothersome. Long periods of sitting or kneeling cause a deep, gnawing ache in the knee.

On examination, there is tenderness just under the medial border of the patella. Flexion and extension of the knee in the supine position produces subpatellar crepitus, and the symptoms of which the patient complains can often be duplicated by pressing the patella firmly down against the femoral condyles while the patient flexes and extends his knee.

Basis of treatment of chondromalacia patellae is elimination of friction between patella and femoral condyles by maintenance of extension of the knee. In many cases, this can be done by a simple explanation of the mechanics of the disability to the patient, who is taught to sit with the knee in full extension and to go up and down stairs with the involved knee straight. Occasionally, a small bandage is applied to the knee simply to remind him of the necessity of maintaining full extension. In the less intelligent or less cooperative patient, a plaster cylinder with the knee in full extension may be necessary. Rarely, surgical planing of the back of the kneecap or complete removal of the patella may become necessary.

A diagnosis of traumatic synovitis was made in only 2 cases in the series of 72. Many more knees than this developed traumatic synovitis, of course, but the recorded diagnosis and administered treatment was, quite properly, that of the underlying ligamentous or meniscal lesion.

Fractures of the patella, unless they are completely undisplaced or consist of very minor

chips, deserve expert care and are usually treated by partial or total patellectomy with suture of the quadriceps tendon and its medial and lateral expansions. Tibial plateau fractures similarly rate expert care, and the disability rates are likely to be high. Tibial spine fractures, usually avulsions by the cruciate ligaments, can occasionally be drawn down into place and fixed with pullout wire loops. Like the other fractures about the knee, they rate high-level specialty care.

Irritation of the medial collateral ligament bursa can present a clinical picture often difficult to distinguish from medial meniscus injury. After a period of repetitive kneeling or crouching work, pain develops along the inner side of the knee and becomes excruciatingly acute if the foot is suddenly externally rotated as when the toe of the shoe is ticked against a chair or table leg. The patient often complains of inability to straighten the knee due to the fact that the swollen and inflamed bursa must squeeze under the leading edge of the medial collateral ligament as the knee is extended.

Examination shows tenderness on the medial joint line just under the leading edge of the medial collateral ligament and very close to the point of maximum tenderness usually found in torn medial meniscus. Careful palpation, however, will usually reveal the bursal sac, and the area of maximum tenderness will often move with the ligament in flexion and extension of the knee. Stressing the knee in the knock-knee position usually aggravates the pain as the fibers of the medial ligament are tightened over the inflamed bursa. Treatment by local injection with corticosteroid has been successful.

Summary

Adequate initial evaluation of the injured knee and determination of definitive treatment lies within the field of responsibility of the industrial physician, the team doctor, and the family physician. Competence in this field stems from a clear knowledge of the mechanics of the joint and the possibilities of damage. With this sound base, a careful history of injury and a patterned examination should, in most cases, yield a confident diagnosis. Many of the knee injuries can be managed by the interested nonspecialist by methods applicable to all joint injuries plus minute attention to quadriceps preservation and rehabilitation.

(The author's address is 277 Alexander St., Rochester, N.Y.)

COLUMBIA UNIVERSITY SCHOOL OF PUBLIC HEALTH
ADMINISTRATIVE MEDICINE PROGRAM OF CONTINUATION EDUCATION

announces

FOURTH ANNUAL INSTITUTE FOR PHYSICIANS IN INDUSTRY

February 3-7, 1961

These institutes are designed to give the industrial physician a period of intensive study in new subject areas which will be of value to him in his practice in the ever-changing field of industrial medical care. Consideration is also given to subjects which are covered in specialty board examinations for the industrial physician. Intensive instruction will be offered in some or all of the following subjects: epidemiology, biostatistics, toxicology, radiation, air pollution, occupational medicine (general review), and applications of insurance in occupational medicine. The Institute is co-sponsored by the American Academy of Occupational Medicine.

For further information inquiries may be directed to the Program of Continuation Education, School of Public Health and Administrative Medicine, 600 West 168th St., New York 32, N.Y.

Industrial Medical Association

THE PRESIDENT'S MESSAGE

ROBERT E. ECKARDT, M.D.

IN MY VISITS to the component societies during the year thus far, a recurring question from the membership has been the reasons for the proposed increase in dues. The simple facts in this situation are that our Association is operating at an annual deficit of about $6,000. Owing to the generosity of our component societies last year, it was possible to reduce our annual dollar deficit (thus the reduction in our operating reserve) to approximately $2,000. Why, the question is asked, has our Association gone from a position of adding annually to the operating reserve, to one of subtracting from it?

There are a number of reasons for this, but an analysis may make our situation a little clearer to our membership. Our income is derived basically from several sources. The major portion is derived from dues, the net amount of which after the component societies have received their 25%, is about $62,000. Another $20,000 is derived from services rendered to OHI and the Central States Society by headquarters plus the management fee and profit from the annual meeting.

Our expenses can be divided into several major categories. Office salaries amount to about $39,000 per year, including travel and retirement pay; journal subscriptions ($6.50 of dues of each member allocated to journal) to about $25,000; and office expenses to about $24,000. The last figure includes rent, light, telephone, supplies, postage, and miscellaneous items, including committee and board of directors expenses.

We are also asked, "How is our journal doing?" Its income is made up of about $25,000 in allocation of dues, advertising revenue of about $37,000, and nonmember subscriptions of $4,500. Its expenses are $47,000 for printing, $20,000 in payroll, advertising representive's commissions and editorial expenses, and $4,000 in postage, telephone, and miscellaneous expenses. The journal thus is close to breaking even, a remarkable feat for so new an offspring.

It is important to note that combined committee, board of directors, and officers expenses to the Association total less than $500, no provision is made for an editor's salary, and there is no provision for printing a directory. We believe our Association should provide funds to defray the expenses of our officers and directors (where this is needed by the man who cannot have his expenses defrayed by his employer), should at the latest by 1962 pay an adequate salary to our editor, should publish an up-to-date directory of our members every one or two years, and should underwrite the expenses of our committees so that they can develop at least the most urgent projects that have been outlined. It is most discouraging to your board, and especially to the committees, that such projects are at a standstill because of lack of funds. Finally, several excellent part-time men have had to decline nominations to our board, thereby failing to qualify as possible future officers, because the expenses of attending our board meetings and other important Association activities could not be met by our Association or from their personal funds. If we are to give real service to our members, these activities are a *must* and the only way we can perform them is through an adequate Association income.

It is also one of our objectives to establish in the headquarters office a comprehensive library devoted to occupational health and industrial medicine and staffed by a competent librarian to enable the office to give prompt and effective service in response to inquiries.

Finally, we have been told that an Association

like ours should have an operating reserve equivalent to 50%-100% of our annual operating expenses. This suggests that we should strive for a $40,000-$80,000 reserve instead of the present $20,000 or less. An increase in dues of $10 per year for active members and fellows will permit us in 1962 to pay a salary to our editor; reissue our directory every one or two years; provide for modest expenses by our committees, officers, and directors; and still add about $5,000 a year to our reserves. We must also make provisions for a replacement for our present managing director (whose retirement is not so far down the road that we can ignore it). These are our requirements. We, your officers and directors, believe that the future of industrial medicine is so vital to us all that you will support us in the proposed $10 dues raise, once you understand its need. Should you need any further information, write me and I shall try to gather it together for you.

May I take this opportunity to wish each member a very merry Christmas, and a most successful and Happy New Year!

Information Please

The questions published in this column are edited versions of requests for information that have been received during recent months. The answers have been prepared by one or more competent authorities in the particular area covered by the question. Readers may address questions to the Editor.

QUESTION: *What are the ill effects, both acute and chronic, of overexposure from going bareheaded in the hot sun (sunstroke and other cerebral manifestations).*

ANSWER: Clinically, we do not differentiate between sunstroke and heat stroke. In either, cerebral manifestations are believed to be the result of general, uncontrolled rise in body temperature to a level of 105°—108° F or more. Heat stroke is more likely during exposure to sun because solar radiation on bare skin can raise the effective environmental temperature by as much as 15° to 20° (F) above prevailing air temperature, increasing the potential thermal load on the nude body by as much as 240 calories per hour. (See Newburgh, L. H., in Physiology of Heat Regulation and the Science of Clothing, Philadelphia, W. B. Saunders Company, 1949, p. 357.) There may also arise, however, heat stroke due to heat production involved in performance of work, due to high humidity, due to high air temperature, or due to a combination of these and direct solar heat.

No special significance is given to solar heating of the head per se in sunstroke. Ordinary clothing will decrease the effective solar load by 50% or more. Covering the head alone may be expected to reduce the over-all solar load by 10% to 20%.

Thus, we adopt the general view that going bareheaded in the sun results in a moderate increase in external heat load; under otherwise difficult conditions this factor may prove critical in producing overstrain in the form of stroke.

QUESTION: *Please discuss the toxicity of Zircon and cite bibliographic sources where this subject is discussed.*

ANSWER: Zircon is the name of the mineral zirconium silicate ($ZrSiO_4$), which occurs naturally in various forms, one of which is transparent and has value as a gem stone. Zircon has some application as a material for refractories and ceramics, but its greatest use would appear to be a mineral source of the metal zirconium.

Zirconium is used as an alloy in steel, cooper, and magnesium, where it imparts certain desirable characteristics. The finely divided metal has been used as a "getter" for residual gases in vacuum tube operations. The pure metal has also found large use in atomic reactor technology.

The silicate, zircon, and the oxide are considered to be inert materials and behave like nuisance dusts in the lungs. The metal dust is also quite inert, although some feel that in high concentrations it may act as a mild respiratory irritant. No cases of harmful effect from exposure to the silicate, oxide, or metal are known to the author. A threshold limit value of 5 mg per cubic meter of air has been proposed and accepted. This refers to the metal and the zirconium content of compounds and is based upon unpublished studies.

Zirconium metal dust and metal sponge are known to react explosively with air under proper conditions. Presumably the dust must be present as an aggregate mass or in high concentration in the air. This threat of explosion would appear to be the greatest hazard in handling the metal.

References

Smyth, H. F., Jr.: Improved Communication—Hygienic Standards for Daily Inhalation, *Indust. Hyg. Quart.* 17:179-180 (June) 1956.

Fairhall, L. T.: Industrial Toxicology, Ed. 2. Baltimore, The William & Wilkins Company, 1957, pp. 135-137.

QUESTION: *What are the appropriate functions of a chiropodist in an industrial medical department? What additional training is recommended for a licensed chiropodist about to be so employed?*

The editorial staff finds it impossible to do justice to this question. Readers with experience in this matter are invited to write to the editor.

Book Reviews

INDUSTRIAL MICROBIOLOGY. Samuel Cate Prescott, Sc.D., and Cecil Gordon Dunn, Ph.D. Ed. 3. Price, $17.50. Pp. 945. McGraw-Hill Book Company, Inc., 330 West 42nd St., New York 36, N. Y., 1959.

The third edition of "Industrial Microbiology" is at once the most advanced and comprehensive treatise on most types of fermentation, and on the respective assays connected with the evaluation of the quality and yield of the final products. Whether leafing through the book or studying it systematically, the reader is struck not only by the organization of the entire work but by the orderly manner in which each process is described. The presentation of methods, although extremely precise, presupposes a sound and accurate knowledge of bacteriology, microbiology, and especially bioengineering. The introduction, which may not be neglected, especially by the newcomer to this field, covers in general terms the multiple uses of microbiological fermentations. These include the production of alcohols, organic acids, acetone, and other end products of value, food manufacturing processes, e.g., cheese, sauerkraut, as well as the retting of fibers in the textile industry. Sterilization, quick-freezing, canning, and other means of arresting fermentation when the latter may become detrimental are usefully discussed.

The outstanding qualities with which the industrially important microbes must be endowed are enumerated:

"1. The ability to grow rapidly in suitable organic substrates and to be easily cultivated in large quantities.

"2. The ability to maintain physiological constancy under these conditions and to produce the necessary enzymes readily and profusely, in order to bring about the desired chemical changes.

"3. The ability to carry out these transformations under comparatively simple and workable modifications of environmental conditions and, since the reactions are exothermic, without the application of large quantities of external energy."

In every process described, the abovementioned fundamental rules have been adhered to.

The book falls logically into three sections, dealing with the yeasts, bacteria, and molds. Each section opens with a chapter on the morphology, classification and general nutritional requirements of the group under consideration.

The actual mechanism of ethyl alcohol fermentation is fully discussed and an interesting account given of the way in which research has proceeded since Lavoisier's investigations in 1789. The brewing industry is dealt with comprehensively. Process flow sheets, analyses of several types of malt beverages and production statistics are included, together with much information on the defects which may be caused in beer by bacteria. The chapters on wine and the distilling industries are perhaps less technical but of great general interest and, as always, better than well documented.

The production of vinegar by bacteria is one of the oldest fermentation industries. The authors describe both slow and rapid methods of manufacture which have been in use for many years. Less widely known is the submerged acetic acid fermentation process developed in 1950 by Hromatka and Ebner, and this is recounted in some detail. The hazards inherent in both types of fermentation and the development of defects in the finished product due either to faulty equipment or to contaminations are summarized.

The account of the production of vitamin B_{12} is one of the brilliant chapters of the third edition. The subject is treated succinctly but completely. Organisms capable of this fermentation include several genera of Streptomyces and many bacteria. This process has been developed rapidly since 1948, and, as usual, the authors have made full use of the information contained in patents taken out on the industrial applications of microbial fermentations. Culture of organisms, production media, conditions of pH, and aeration are all described. Perhaps more space could have been devoted to the recovery of the vitamin from the fermentation broth and its final purification using either solvent, solvent extraction or column separation, yet adequate references are given. The current assay methods are compared.

The production of amino acids by microorganisms has evoked much interest. The patented process for the manufacture of L-lysine from a glycerol-corn steep liquor medium via diaminopimelic acid is described. This process makes use of such organisms as Escherichia coli, Corynebacterium diphtheriae, and Mycobacterium tuberculosum. L-Glutamic acid can be obtained from wheat gluten, soybean cake, or, as in Japan, sweet potatoes. The biochemistry of these reactions is authoritatively discussed.

This is a book which deals with its subject in the round. Information on the history and uses of a product are given, together with details of the fermentation process, organism, choice of media, operating conditions, and equipment. The biochemistry of the process under consideration is always most clearly discussed. Many methods of industrial preparation are described at length and others summarized, but more than adequate references for all stages of the process are cited. The index is accurate, well arranged, and complete. The typographical layout is excellent, and tables and diagrams have been most carefully designed. The authors' lucid style combines with the very interesting material presented to make this book the definitive work on the practical applications of

microbiological fermentations while at the same time insuring highly pleasurable reading.

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A PSYCHIATRIST'S WORLD: THE SELECTED PAPERS OF KARL MENNINGER, M.D. Edited by Bernard H. Hall, M.D., with a foreword by Marion E. Kenworthy, M.D. Price, $10.00. Pp. 931. The Viking Press, 625 Madison Ave., New York 22, N. Y., 1959.

The world of Dr. Karl Menninger has a breadth and enthusiasm of rare quality. In this expertly edited and annotated volume are assembled 83 articles published between 1919 and 1959 which amply document the contributions of Dr. Menninger to psychiatry. This volume clearly documents the well-earned professional reputation of the author of "Man Against Himself," "The Human Mind," and "Love Against Hate."

Dr. Bernard Hall, in his introduction, says: "Karl Menninger was not content with his accomplishments as a leader in psychoanalytical education. He foresaw that many physicians would not become psychoanalysts and that the greatest contribution of psychoanalysis would be not through its use as a treatment method but through the application of its principles about human behavior."

For even the casual reader of "A Psychiatrist's World," a fresh understanding of people, well or sick, develops on every page. In stimulating, readable prose, these papers are divided into six parts: "The Man," "The Clinician," "The Theorist," "The Teacher," "The Psychiatrist Afield" and "The Historian of Psychiatry."

Of particular interest to the occupational physician will probably be the case studies of self-destructiveness, of psychosomatic syndromes, and of diagnostic and treatment methods.

But this suggestion may be presumptive, since the topics range from "Men, Women and Hate" to "Citizens and Schools," from "Psychology of a Certain Type of Malingering" to "One View of the Kinsey Report," and, from "The Meaning of the Hospital" to "The Doctor as a Leader."

This reviewer found Part II: The Clinician to be the most rewarding papers in the volume. In somewhat more than 300 pages one finds the equivalent of a textbook in clinical psychiatry. While most papers in this section represent studies in the earlier psychiatric career of Dr. Menninger, the roots of his later thinking are in clear evidence. Most physicians will find ready identification with his early organic orientation. Most will also move with him to the more dynamic, optimistic, hopeful clinical approach so evident in his more recent works. In following this gradual shift in pattern of approach to psychopathology, Dr. Menninger carries the reader with him toward acceptance of the usefulness of psychoanalytical concepts in psychiatry.

To read "A Psychiatrist's World" is a liberal education, a rewarding experience—one which should not be missed.

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EMERGENCY WAR SURGERY. U.S. Armed Forces Issue of NATO Handbook. Prepared for use by the Medical Services of NATO Nations. Price, $2.25. Pp. 411. United States Department of Defense, 1958. Available from the Superintendent of Documents, Government Printing Office, Washington 25, D. C.

As Frank B. Berry, M.D., Assistant Secretary of Defense, notes in the forward, this book was prepared for use by the medical services of the NATO Nations and represents the experience and thinking of the foremost military medical authorities of all the NATO Nations, but primarily those of France, the United Kingdom, and the United States. Being indeed a handbook, it is of necessity most didactic but also most authoritative. It follows, then, that detailed discussions and explanations of the methods of treatment recommended cannot be contained within its 365 text pages. One feels that all phases of combat zone medical care are covered, at least in outline form.

After a brief discussion concerning the mechanism of wound production by high velocity missiles, the authors discuss such basic conditions as thermal burns, crush injuries, blast injuries, muscle injuries, shock, resuscitation, and metabolic disturbances. One entire chapter is devoted to that most important function of the military medical man—the sorting of casualties. This problem is discussed as seen at all echelons of medical care and as it particularly applies to the various types of injuries. Special emphasis is placed upon this function in the treatment of mass casualties. Having outlined broadly the care to be given at the Battalion Aid Station, the Division Clearing Station, and the Evacuation Hospital, the work goes on to separate chapters discussing each of the types of injury broken down by specialty; that is, a consideration of vascular injuries, soft-tissue injuries, abdominal injuries, neurosurgical injuries, orthopedic injuries, amputation, maxillofacial injuries, eye injuries, and the others. Each of these chapters concludes with at least a paragraph devoted to the considerations of how this group of patients would be handled in the event one were dealing with mass casualties. This concept, of course, is foreign to any held by the usual civilian physicians but can be extremely important in any military operation or civilian disaster and is to be anticipated for both the military and civilian components in the event of thermonuclear warfare.

This publication would be of little value as a reference work and is certainly not a textbook. It is, on the other hand, a compact handbook prepared by world authorities and based upon the all too

great volume of empirical experience gathered from World Wars I and II and the Korean police action. It will prove to be of tremendous value to the physician who suddenly finds himself responsible for the care of patients with this type of injury. It will be of special value in guiding the forward surgeon in the care of casualties whose injuries are outside his own field of experience.

—ROBERT C. COMBS, M.D.
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MEDICAL DEPARTMENT, UNITED STATES ARMY, SURGERY IN WORLD WAR II: NEUROSURGERY, Vol. I. Prepared and published under the direction of Major Gen. S. B. Hays, The Surgeon General, United States Army, Price, $5.00. Pp. 466. Office of the Surgeon General, Department of the Army, 1958. For sale by the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.

Volume I of Neurosurgery in World War II is divided in two parts. Part One deals with the history and administrative history of wartime neurosurgery and the administrative problems and plans of development of military neurosurgery. The organizational planning necessary for the establishment of efficient neurosurgical service in both the Mediterranean and European theaters is described in a fascinating manner.

Part Two consists of 12 chapters dealing mainly with open head injuries. The closed head injuries are discussed briefly, while the blast-concussion and cerebral injuries are described in greater detail. Considerable emphasis is placed on the various techniques used and the various materials tried in the repair of skull defects. Tantalum was considered to be the most satisfactory. (The reviewer, who used tantalum exclusively in the Naval Hospitals, during the war has since given up its use and is now using acrylic or autogenous bone as being more satisfactory.) Penetrating wounds of the brain and ventricles are well discussed in a definitive manner, with the conclusion that prompt debridement of the entire missile tract, including the ventricles, is necessary for penetrating wounds of the venticles and that antibiotic therapy is effective only when combined with definitive surgery. The chapter on Post-Traumatic Epilepsy by Walker is particularly good, with an excellent discussion of the diagnosis, case selections, surgical requirements, and techniques. Likewise, the chapter on Speech Disorders resulting from gunshot wounds in the head and neck, describing the various types, causes, and treatments, is worthy of careful reading. The concluding chapter on the clinical and pathologic aspects of fatal missile-produced craniocerebral injuries, with the pathology well illustrated by both description and illustration, should provoke detailed study by anyone interested in neurophysiology and neuropathology.

In summary, Volume I of Neurosurgery in World War II constitutes an excellent collection of monographs by leading neurosurgeons, who had the ability to observe, describe, and report.

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Publications Available

The following list of publications has been compiled on the basis of probable interest to the readers of this journal and is presented solely for the purpose of giving notice of their availability.

Film Reference Guide for Medicine and Allied Sciences. Interdepartmental Committee on Medical Training Aids (ICMTA). Price, $1.00. Pp. 199, with introduction and instructions, 1960. Available from the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.

This is a catalogue of the visual aids available for use in teaching health personnel. It was developed to meet the need for such information by various governmental agencies. ICMTA comprises a representative of the Air Force, Army, Navy, Armed Forces Institute of Pathology, Public Health Service, and Veterans' Administration. The criterion for listing is that a film must be useful to one of the participating agencies. The Guide is printed annually, each new issue superseding the previous one.

There is a short instruction on how to use the Guide, followed by information on how to obtain films. This is followed by an alphabetical list of film distributors with mailing addresses. A list of subject categories, arranged alphabetically, shows the pages on which respective categories begin. The main body of the Guide is a list of some 2,000 films by exact title, a brief description of the material covered, and the distributor. All of these are arranged within categories. The Guide closes with an alphabetical listing of all films by title, disregarding subject category.

Occupational Health Hazards, Their Evaluation and Control. Bulletin 198. Prepared by William G. Griffin and Andrew D. Hosey. Price, $0.25. Washington, D.C., U.S. Department of Labor, Bureau of Labor Standards, 1958. Replaces Bulletin 83, by R. P. Blake, published in 1947 under the Title Outline of Occupational Disease Control Through Engineering. For Sale by the Superintendent of Documents, U.S. Government Printing Office, Washington 25, D.C.

This short (32 page) booklet is a primer in the background and methodology of industrial hygiene. Four chapters describe the basic factors, starting with a classical catalogue of the commonly encountered sources of occupational health hazards. A brief description is then given of the manner in which the worker is exposed to these noxious influences, the methods of appraisal and the usual procedures of environmental control.

Editorials

The Deaf at Work

A PART of the experience at Oak Ridge, Tenn., during the war years was a necessary accommodation to community services of varying quality. The Manhattan Project was new, vast, and urgent; and some of its work had to be carried on with the residue of the country's labor force. By contrast with the city's norm for services, we had available an impressively efficient laundry. The manager of this institution, it turned out, had discovered how to get employees who were not "from the bottom of the barrel." He had staffed his plant with trained workers who were deaf.

Now, some 15 years later, there comes to hand a report of a study dealing in broad terms with the matter of deafness and employment.* Laundry work is still a prominent occupation among those who earn a living as trained operatives. But exceeding this by an almost 3 to 1 ratio is employment in the printing crafts. Skilled jobs that require a few years to learn—machine operations and crafts of various kinds—are generally those sought and successfully filled by the deaf. Relatively few become managers or clerks, and still fewer end up as laborers. Lipreading and writing are generally used in communications with fellow workers who can hear and manual signs with other deaf persons.

Most of those questioned in the study believed that deafness is a handicap that precludes advancement in industry beyond the job for which they were specifically trained. Despite this, they are well pleased with their jobs and their employers. Refreshing news in these uneasy times is that only 1% of those questioned believed that there is an "unfriendly" attitude on the part of workers who can hear, and 85% rated their working conditions as "good."

That this may not be an absolutely true picture is recognized by the authors of the report, who point out the shortcomings of their study. Its design and analysis were accomplished at Gallaudet College, and with these aspects there is little wrong. There is some weakness in the way the basic data were collected by the National Association of the Deaf, to whom this portion of the study was delegated. Under a chairman in each state, local volunteer interviewers worked among the deaf known to them or known to others who were deaf, using religious or social centers of those so handicapped for further contacts. A "sample" of some 10,000 persons so interviewed was unduly weighted in favor of white persons between the ages of 20 and 60 years. Despite these drawbacks, the study gives us a partial understanding of how deaf persons fit themselves into the industrial structure.

Apparently the agencies created to educate the deaf carry out their mission creditably; the social climate for persons with this affliction is not hostile; and the deaf accomplish a great deal for themselves through their own groups. Taken all together, this is not a discouraging story.

*Lunde, A. S., and Bigman, S. K.: Occupational Conditions Among the Deaf, Washington, D.C., Gallaudet College, 1959.

Another Year

SOME ARE still alive who remember the relieved comment with which workmen closed the day: "Another day, another dollar." For our JOURNAL, another year has meant more than another volume. The insecurities of beginning are behind us, and we have taken on some of the characteristics of an established enterprise.

We believe that a good share of the significant work in industrial medicine in America comes to our journal unsolicited. (We still chase after the rest.) An increasing number of good articles from other countries are coming our way. A system of communication has been developed with IMA Component Societies that keeps us reasonably well apprized of local program items which ought to be published.

Looking back, things look good. Up ahead, they look even better. The strength of our JOURNAL is in the abilities and energy of literally hundreds of Association members and friends who are determined to make it great. The time is at hand for a well-earned pause and a cordial wish all around for a Merry Christmas and a Happy New Year.

MISSING PAGES 609-610 ARE
COVER PAGES TO THE INDEX.

Index to Volume II

January through December, 1960

This index consists of three parts, a subject index, an authors index, and a general section index. The parenthetical matter following the page numbers indicates the month of issue. The abbreviations are: January (Ja); February (Fe); March (Mr); April (Ap); May (My); June (Je); July (Jl); August (Ag); September (S); October (O); November (N); December (D).

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About the Authors

► PAUL GROSS, M.D., is senior author of the article in this issue, "Tuberculosilicosis: A Study of its Synergistic Mechanism." This is the latest of a series of publications by the group headed by Dr. Gross at the Research Laboratory of the Industrial Hygiene Foundation, Mellon Institute, Pittsburgh. This group has for some years been engaged on fundamental studies of the mechanisms of pulmonary damage by industrial materials.

Dr. Gross is an educational product of Cleveland, Ohio. He is a graduate of East Technical High School of that city, and holds A.B. and M.D. degrees from Western Reserve University. After a year of internship at Charity Hospital, he returned to Western Reserve for graduate work as a George Washington Crile Research Fellow in Pathology, and received the M.A. degree in 1929. He was a resident in pathology at City Hospital for the years 1929-1931. This was followed by a full year as an assistant to Professer Erdheim in Vienna.

Dr. Gross' interests over the years have gradually and consistently moved from the practice of pathology to research in the pathogenesis of pulmonary disease. After four years as pathologist to St. Vincent's Charity Hospital in Cleveland he moved to Pittsburgh, where he was pathologist to the Hospital of Western Pennsylvania and, later, St. Joseph's Hospital.

Dr. Gross' affiliation with the Industrial Hygiene Foundation, begun on a part-time basis is 1948, has occupied an increasing fraction of his time. He recently became Adjunct Professor of Industrial Disease Pathology in the Department of Occupational Health in the Graduate School of Public Health, the University of Pittsburgh. He is a member of IMA, American College of Physicians, and various specialty organizations of pathologist.

► MARIAN L. WESTRICK, PH.D., is a co-author of the article in this issue, "Tuberculosilicosis: A Study of its Synergistic Mechanism." Miss Westrick is a graduate of Mt. Aloysius Academy, Cresson, Pa. and has received the B.S., M.S., and Ph.D. degrees from the University of Pittsburgh. She has been engaged in research toxicology at the Laboratory of the Industrial Hygiene Foundation, Mellon Institute, Pittsburgh, since 1951.

► JAMES M. MCNERNEY, Jr., M.P.H., is Assistant Research Toxicologist at the Laboratory of the Industrial Hygiene Foundation, Mellon Institute, Pittsburgh. Mr. McNerney holds three degrees from the University of Pittsburgh: B.S., M. Litt., and M.P.H. He is a co-author of the article in this issue, "Tuberculosilicosis: A Study of its Synergistic Mechanism."

► STANLEY GREENHILL, M.D., was born in Glasgow, Scotland, and was educated at the Universities of Glasgow, California, and Toronto. During the War, he served with the Royal Air Force. He returned to Canada at the end of hostilities.

Dr. Greenhill received both his M.D. and his D.P.H. from the University of Toronto. Upon completion of further postgraduate training, he obtained his certification in internal medicine from the Royal College of Physicians, Ottawa. He is a Fellow of the American College of Physicians, a Fellow of the Industrial Medical Association, and a Member of the Royal Society of Health, London, England. At the University of Alberta, he is a lecturer in the department of medicine and contributes to the teachings in other faculties. In September, 1959, he was appointed Professor and Head of the Department of Social and Preventive Medicine, University of Alberta.

Dr. Greenhill is author of the article "Mental Health and the Worker," in this issue.

► MALCOLM H. MERRILL, M.D., M.P.H., is a career officer in the California State Department of Public Health. He joined the department as Chief, Bureau of Venereal Diseases, in 1937. Four years later he became Chief of the Di-

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vision of Laboratories which post he held until 1954. During most of these years he served also as Deputy Director of the Department, and, for a brief period, as Acting Director. He became Director in 1954.

DR. MERRILL graduated from Utah State Agricultural College in 1925 with a B.S. degree in chemistry. He then went to St. Louis University as a graduate student in bacteriology, receiving the M.S. degree in that field in1927. He stayed on as an assistant in bacteriology while attending medical school until he received the M.D. degree in 1932. Graduation was followed by three years of work as an assistant at Rockefeller Institute for Medical Research and a year of internship and a year of residency in medicine at the University of California Hospital. He received the M.P.H. degree from the University of California in 1946.

DR. MERRILL was President of the American Public Health Association in the term just closed, 1959-1960. He has served on many advisory committees to the U.S. Public Health Service, and he was a member of the U.S. Public Health Mission to Russia in 1957. His honorary societies include Alpha Omega Alpha, Delta Omega, and Phi Kappa Phi. He is the author of some 40 publications in scientific journals in the fields of bacteriology, immunology, serology, and public health administration. His article in this issue of our journal is "Air Quality for California Communities."

► F. W. WILSON, M.D., is author of "Problems in Industrial Cardiology," in this issue. Dr. Wilson is a full time member of the medical staff at the large refinery of Texaco, Inc., located at Port Arthur Texas. His work there is limited to the internal medicine aspects of industrial practice, with more than half of his time devoted to cardiology problems. This is his thirteenth published article in cardiology and the general field of internal medicine.

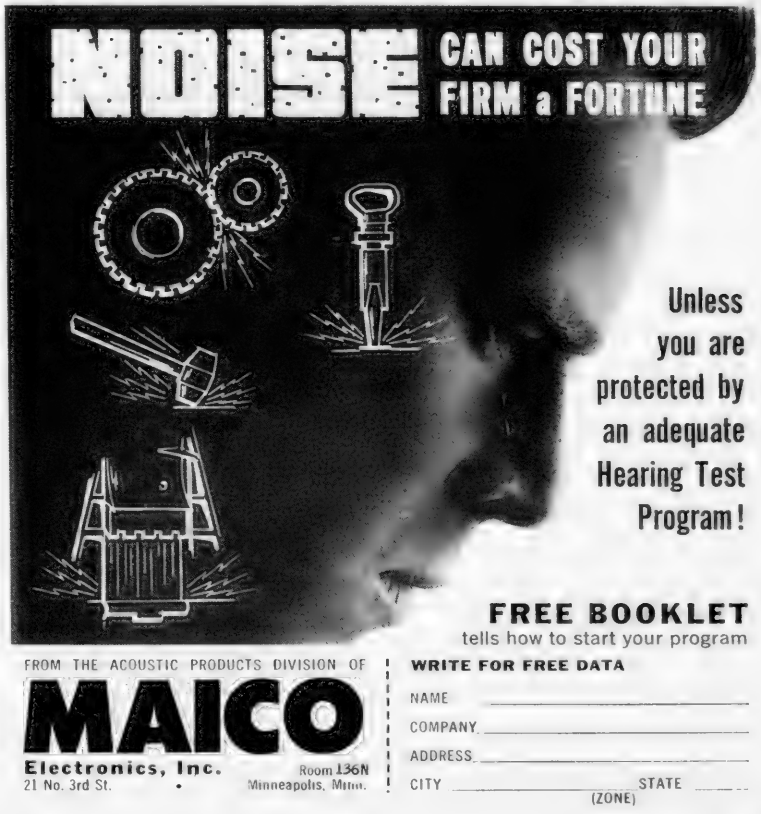
Dr. Wilson, a native of Texas, received the B.S. degree from Texas A & M College in 1935 and the M.D. degree from Baylor University in 1940. After an internship at Robert B. Green Hospital, San Antonio, he served a residency in internal medicine at Southwestern Medical College, Dallas. He is a veteran of World War II and served during the Korean conflict with the rank of lieutenant colonel.

Dr. Wilson is a member of IMA, a director of the Texas IMA, and a member of the Committee on Occupational Health of the Texas Medical Association. He is certified by the American Board of Internal Medicine, a Fellow of the American College of Chest Physicians, and a member of the Texas Academy of Internal Medicine. He was for some time a clinical assistant professor of internal medicine in the Postgraduate School of Medicine of the University of Texas.

► HARRY C. HALSTED, M.D., is author of the topical review in this issue, "Industrial Methemoglobinemia." This has been developed from a background of case experience with the phenomenon during almost a decade of experience at the American Cyanamid Company's plant at Bound Brook, N.J.

Dr. Halsted received a B.A. degree from Columbia (University) College, New York City, in 1937. He then attended the University of Vermont College of Medicine, Burlington, Vt., graduating with the M.D. degree in 1941. After a rotating internship at Flower and Fifth Avenue Hospitals, New York City, he entered the U.S. Army for four years of service in its Medical Corps. After the war he returned to Flower and Fifth Avenue Hospitals for a two-year residency in otolaryngology, following which he practiced in that specialty for two years.

Dr. Halsted joined the medical staff of the Cyanamid plant at Bound Brook in 1950. About four years later he became chief of that staff, and in 1958 he was promoted to Assistant Director of Employee Health, with offices at the firm's headquarters in New York City. He is certified in occupational medicine by the American Board of Preventive Medicine and holds memberships in the American Medical Association and in IMA. —EDITOR



People and Events

► EDWARD V. HENSON, M.D., joined Standard Oil Company (Indiana) on December 1 as Medical Research Associate, it was announced by GILBEART H. COLLINGS, M.D., Medical Director. DR. HENSON is in charge of toxicology research and is located at the company's headquarters in Chicago.

Prior to his present appointment DR. HENSON was Medical Director for Union Carbide Chemicals Company, South Charleston, W. Va. He attended Pennsylvania State University and received a B.S. degree in chemistry in 1939. He obtained his M.D. degree from Jefferson Medical College in 1943 and served his internship at St. Francis Hospital in Charleston, W. Va. He joined Union Carbide in 1947 as a staff physician and became head of the department in 1950.

DR. HENSON is Assistant Editor of the JOURNAL OF OCCUPATIONAL MEDICINE and a Fellow of the Industrial Medical Association, a member of the American Industrial Hygiene Association, the American Public Health Association, and is certified in occupational medicine by the American Board of Preventive Medicine.

Edward V. Henson, M.D.

Theodore E. Allen, M.D.

► THEODORE E. ALLEN, M.D., has been appointed Medical Director for Mobil Oil Company, an operating division of Socony Mobil Oil Company, Inc., covering the United States and Canada. He continues to serve as Regional Medical Director in the East and as Associate Medical Director of Socony Mobil.

DR. ALLEN is a graduate of Williams College and received his M.D. degree from Yale University School of Medicine in 1940. He had graduate medical training at the School of Tropical and Military Medicine of the U.S. Army and earned the Master of Public Health degree at Harvard in 1948.

His memberships in professional societies include the American Academy of Occupational Medicine, the American Medical Association, the Industrial Medical Association, the Royal Society of Tropical Medicine and Hygiene and the American College of Preventive Medicine. He is certified in public health by the American Board of Preventive Medicine.

► ROY W. MONEY, M.D., has been appointed Chief Physician at the Nashville Glass Plant of the Ford Motor Company.

DR. MONEY was graduated from Tennessee Polytechnic Institute, attended Memphis State College, and received his M.D. degree from the University of Tennessee College of Medicine in 1944. He served a rotating internship at the Nashville General Hospital. In the last six

PRELIMINARY ANNOUNCEMENT

Post-Conference Tour of Hawaii

Following the Industrial Health Conference April 11, 12, 13, 1961, in Los Angeles, there will be an official IHC tour to Honolulu and some of the Hawaiian Islands. The basic tour will be 10 days, air travel each way—or 15 days, go by air, return by ship—with optional additional arrangements. An experienced travel organization has been appointed as our official tour representative, and we are now discussing details with them and our component society, the Hawaii Association of Plantation Physicians. An official tour brochure will be prepared for distribution later. In the meantime, it would be helpful if any who may be interested in the trip would complete and return attached coupon. There is no obligation.

To: Clark Bridges, Managing Director
Industrial Health Conference
28 East Jackson Boulevard
Chicago 4, Illinois

We would like more information about the tour to Hawaii following the 1961 Industrial Health Conference. Please send details as soon as available.

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Planning Committee for the 1961 American Industrial Health Conference at its meeting November 5 at the Biltmore Hotel, Los Angeles. Committee chairmen reported progress in several areas, including some new approaches to scientific sessions. Preliminary programs for the Conference are to be ready for mailing early in January.

years he has completed postgraduate courses in radiology, electrocardiography, internal medicine, fractures and dislocations and ophthalmology.

Prior to joining Ford Motor Company, DR. MONEY spent 12 years in general practice at Pulaski, Tenn. During this time he was a member of the Giles County Hospital staff. From 1945 to 1947 he served with the Medical Corps of the U.S. Army which included duty with various Army hospitals and service in the Philippines. DR. MONEY is a member of the Industrial Medical Association, and is secretary of the Giles County Medical Society.

► C. HUNTER MONTGOMERY, M.D., has joined the Medical Staff of the Humble Division, Humble Oil and Refining Company, Houston, Tex., to serve the Medical Division in the field of internal medicine. He was in private practice prior to his association with Humble.

DR. MONTGOMERY did his undergraduate work at Lynchburg College, Lynchburg, Va., and received his medical training at the University of Virginia School of Medicine. Following graduation in 1953, he served an internship and two years of medical residency at the University of Virginia Hospital, completing his medical residency at Baylor University College of Medicine in 1958. DR. MONTGOMERY served for two-and-a-half years with the United States Public Health Service, where he was assigned to the Communicable Disease Center.

► AMERICAN INDUSTRIAL HEALTH CONFERENCE is to be the new name of the Industrial Health Conference. This was accomplished by action of the Board of Directors of the Industrial Medical Association and approved by the Joint Planning Committee representing the participating groups. The change was made to distinquish this national meeting from local and state industrial health conferences.

Nominations Open

The health Achievement in Industry Award Committee of the Industrial Medical Association requests the membership to submit the names of companies thought to qualify for its award.

Candidates for this award shall be considered on the basis of excellence of occupational health program; imagination by which the program is conceived; dynamic manner in which the program is executed; active participation of management in the program and its tenets; and the general participation of the employees.

Winners of this award for the last six years are as follows: 1960, General Motors Corporation; 1959, Kaiser Steel Company; 1958, Ford Motor Company; 1957, Humble Oil Company; 1956, Pennsylvania Railroad; 1955, Sonoco Products.

Supporting information must accompany nominations. Since this committee must make its selection by the end of the year, it is imperative that nominations be forwarded immediately to Richard A. Sutter, M.D., Chairman, Sutter Clinic, 819 Locust St., St. Louis 1, Mo.

► JAMES A. RUTHERFORD, M.D., has recently joined the medical staff of General Electric Company's Hanford Atomic Products Operation in Richland, Wash.

DR. RUTHERFORD received his B.A. degree from New York University. From 1943 to 1946 he served in the U.S. Naval Reserve as Engineering Officer in the Epidemiology and Malaria Control Unit. He then returned to New York for his medical training, receiving his M.D. degree from State University of New York College of Medicine in 1950.

He served his internship at Queens Hospital in Honolulu and spent the next five years as industrial physician for three sugar companies in Hawaii. During 1956 DR. RUTHERFORD served as Medical Officer for the Mare Island Naval Shipyard, Vallejo, Calif. From 1956 until his present appointment he was Medical Officer at Pearl Harbor Naval Shipyard, Oahu, Hawaii. He is a member of the Industrial Medical Association, the Hawaii Medical Association and the American Medical Association.

People and Events

► DAVID TINGLE, M.D., has been appointed District Medical Director for the Frick Coal District, U.S. Steel Corporation, Uniontown, Pa. For the last two years he has been studying under a Research Fellowship at Johns Hopkins Hospital School of Public Health, and recently completed his course of study leading to a Doctorate in Public Health.

DR. TINGLE was graduated from the University of Sheffield Medical School, Yorkshire, England, in 1952. He received a Diploma in Public Health and in Industrial Health from the London School of Hygiene in 1956, and served his internship at the Sheffield Royal Infirmary and Western Hospital, Doncaster, both in Yorkshire, England. He was a Medical Officer in the Royal Air Force from 1953 to 1954. Following his release from the service he was in general industrial medical practice in Sheffield, Yorkshire and Dagenham, Essex, England. In 1957 he came to the United States and served as the County Health Officer in Elizabethtown, North Carolina for one year.

Rupert C. Burtan, M.D.

► RUPERT C. BURTAN, M.D., has been appointed Plant Physician for the B. F. Goodrich Footwear and Flooring Company, Watertown, Mass. For the last two years he has served as Senior Assistant Surgeon with the U.S. Public Health Service in Honduras as a part of the Point Four Technical Aid Program.

DR. BURTAN received his M.D. degree from the College of Physicians and Surgeons, Columbia University in 1947 and served his internship at the Lenox Hill Hospital in New York. He spent the next three years in Aruba, Netherlands West Indies, with the Lago Oil and Transport Company, then a year with North Atlantic Constructors in Greenland. DR. BURTAN then returned to Columbia for postgraduate training in public health and earned the masters degree in 1955. He is currently preparing his Ph.D. thesis in the same field. He is a member of the Industrial Medical Association, the Massachusetts Medical Society and the American Medical Association.

► LEON J. WARSHAW, M.D., Consultant in Occupational Health, and Medical Director for United Artists Corporation and Paramount Pictures Corporation, was recently in London to guide the organization of an industrial medical service to serve the British employees and staff of United Artists Corporation and Columbia Pictures Corporation. This will be the first cooperative

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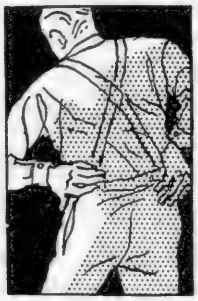

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unit serving several small plants staffed by white-collar personnel in England. At the invitation of DR. RICHARD SCHILLING, Professor of Occupational Medicine at the London School of Medicine, DR. WARSHAW lectured on "Recent Developments in Occupational Health Programs in the United States."

► HOWARD B. HAMILTON, M.D., has been appointed Medical Officer for the Pennsylvania Railroad's Southwestern Region with offices in Indianapolis. He succeeds the late N. J. FINE, M.D.

DR. HAMILTON has been with Pennsylvania Railroad for 20 years, having served as Medical Officer in Wilmington, Del., for the last five years. He is a graduate of the University of Chicago and received his M.D. degree from Rush Medical College in 1936. He was in private practice in Gillette, Wyoming and Chicago prior to joining the Pennsylvania Railroad.

► Industrial Medical Association of Philadelphia elected the following officers and directors at a meeting on October 26: President—HOWARD J. JOHNSON, JR., M.D.; President-Elect—BERNARD BEHREND, M.D.; Secretary—EMILIE SUZANNE LOEFFLER, M.D.; Treasurer—JAMES F. HAYS, M.D.; Directors—GLENN S. EVERTS, M.D.; WILLIAM A. TOMASCO, M.D., and JACK R. ZAHN, M.D.

► Western Ohio Industrial Medical Association held its first annual meeting at the National Cash Register Company, Dayton, on September 14. The meeting was preceded by an afternoon tour of the plant. The scientific portion of the meeting was held in the company's main dining room with approximately 50 physician's in attendance.

After a brief welcome by WALTER BOSWELL, NCR's Vice- President of Industrial Relations, the business session was conducted by President JACK L. HARRIS, M.D., Medical Director, Armco Steel Corporation.

H. W. LAWRENCE, M.D., Medical Director of Procter and Gamble Company and past president of the Industrial Medical Association, presented a short discussion of the development of industrial medicine in the Ohio area.

The scientific portion of the program was presented by CAPT. ALVIN HYDE, a research physician at the Air Force Research and Development Center, Wright-Patterson Air Force Base. DR. HYDE'S discussion concerned the physiological aspects of horizontal acceleration as this relates to manned space flight. The research and development effort along space medicine lines at Wright-Patterson Air Force Base, plays a significant part in determining the ability of a human to survive and withstand the

Announcement

Applications are being solicited for Scientific Exhibits for the 1961 Industrial Health Conference which will be held in Los Angeles. Exhibits will be shown April 11 to 13. Application blanks and information may be obtained from HOMER E. ELMQUIST, M.D., Chairman of Scientific Exhibits Committee, 629 S. Westlake Ave., Los Angeles 57, Calif.

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People and Events

rigors of space exploration. DR. HYDE expressed the need for further research and development on various responses of the human organism to stress situations and metabolic demands of space travel.

► New Jersey Industrial Health Conference was held October 22 at Far Hills Inn in Somerville, N. J. The meeting, which was attended by 415 persons, was sponsored by the New Jersey Industrial Nurses Association, the Industrial Medical Association of New Jersey, and the New Jersey Section of the American Industrial Hygiene Association.

Highlight of the meeting was COL. GEORGE M. KNAUF'S presentation of color movies showing the missile launchings and failures at Cape Canaveral. COLONEL KNAUF is attached to the 6550th USAF Hospital, Patrick Air Force Base, Florida.

Other speakers and their topics included: ROBERT R. BLAKE, PH.D., Department of Psychology, University of Texas—"Power—the Achilles Heel of Modern Organizations"; FRANK T. KOCH, Director of Public Relations, Ciba Pharmaceutical Products, Inc.—"Prescription Drugs and Your Pocketbook"; CHARLES A. HERRMANN, Director, New York District, Food and Drug Administration—"New Forms of Consumer Protection"; DR. BERNARD L. OSER, President and Director, Food and Drug Research Laboratories, Inc.—"Industry's Problems in Adjusting to New Food Laws"; LT. DOLORES B. O'HARA, USAF, NC, 6550th USAF Hospital, Patrick Air Force Base, Florira—"Pre-Flight Feeding of the Astronaut."

► Industrial Medical Association of Pittsburgh held its sixth annual meeting and scientific session at Mellon Institute on September 30 with 115 physicians, nurses, and industrial hygiene engineers gathered for the day-long program.

Welcoming the group, the Hon. Joseph. M. Barr, Mayor of Pittsburgh, described the basic philosophies leading to the renaissance of Pittsburgh and emphasized the cooperation between industry, the public and state and local governments in its achievement.

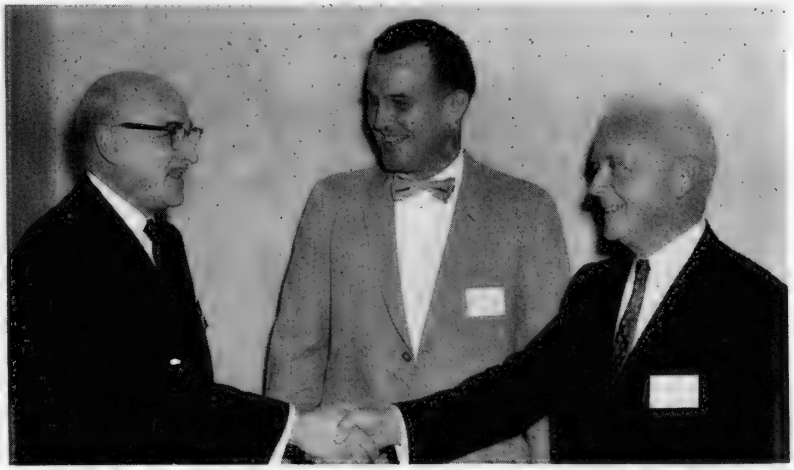
The morning program consisted of a panel discussion on "Legal Aspects of Industrial Medical Practice" with Paul A. Davis, M.D., IMA Counselor for Eastern Ohio, as Moderator. Participants were William A. Challener, Jr., General Counsel for the Allegheny Medical Society; A. J. Blair, M.D., Medical Director of the Consolidated Natural Gas Companies, and IMA Counselor for Eastern Pennsylvania; and Herman J. Pocock, M.D., Physician at the Lamp Division of General Electric Company. The presentations by the panel members were followed by a question and answer period which elicited discussion on many problems involving the status of the nurse in industry, legal responsibilities of the physician, and workmen's compensation.

In the afternoon session, Kenneth M. Morse, Director of Industrial Hygiene, United States Steel

Wanted

The January, March and April, 1960, issues of the Journal of Occupational Medicine are in short supply. Anyone having extra copies of these issues is requested to send them to the Publication Office, Journal of Occupational Medicine, 28 East Jackson Blvd., Chicago 4, Ill. The three issues may be sent in one envelope via second class transient rate for 27c.

Members of a panel on "Legal Aspects of Industrial Medical Practice" at the sixth annual meeting of the Industrial Medical Association of Pittsburgh, left to right: A. J. Blair, M.D., William A. Challener, Jr., and Herman J. Pocock, M.D.



Officers of the Industrial Medical Association of Pittsburgh, left to right: Daniel C. Braun, M.D., newly elected President; Merle Bundy, M.D., Secretary-Treasurer; and Daniel V. Dougherty, M.D., outgoing president.

Corporation, outlined the "Role of Industrial Hygiene in Industrial Medicine," stressing the increasing importance of environmental hygiene in the light of expanding health consciousness on the part of the public and the extension of environmental hazards to the home through do-it-yourself projects. The second afternoon paper was presented by William L. White, M.D., Associate Professor of Surgery and Head of the Department of Plastic Surgery, University of Pittsburgh, who spoke on "A Program for Management of Hand Injuries."

Following the scientific meeting, the business meeting was held and Fellowship certificates presented to George Donnelly, M.D., Paul Mesaros, M.D., and E. R. Plunkett, M.D. The constitution was amended to permit a change of name to the Industrial Medical Association of the Pittsburgh-Cleveland Area.

► New England Industrial Medical Association, Inc., held its annual meeting on November 2 at which time the following officers and directors were elected: President—HARRY R. MUSHLIN, M.D.; Vice-President—JOHN F. KILGUS, JR., M.D.; Secretary-Treasurer—JAMES I. ROBERTS, M.D.; directors—ROGER E. ALLEN, M.D.; JOHN J. FINN, JR., M.D.; ROBERT GOLDSON, M.D.; STANLEY SPRAGUE, M.D.; JOSEPH H. KAPLAN, M.D.; and C. F. YEAGER, M.D.

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Statement of Ownership

Statement required by the act of August 24, 1912, as amended by the acts of March 3, 1933, July 2, 1946, and June 11, 1960 (74 Stat. 208) showing the ownership, management, and circulation of Journal of Occupational Medicine, published monthly at Chicago, Illinois, for October 1, 1960.

1. The names and addresses of the publisher, editor, managing editor, and business managers are: Publisher, Industrial Medical Association, Inc., 28 E. Jackson Blvd., Chicago 4, Ill.; Editor, A. G. Kammer, M.D., 2057 Beechwood Blvd., Pittsburgh 17, Pa.; Managing Editor, none; Business Manager, Clark D. Bridges, 28 E. Jackson Blvd., Chicago 4, Ill.

2. The owner is: Industrial Medical Association, Inc., 28 E. Jackson Blvd., Chicago 4, Ill., a voluntary association of physicians incorporated not-for-profit in the State of Illinois. There are no stockholders.

3. The known bondholders, mortgagees, and other security holders owning or holding 1 percent or more of total amount of bonds, mortgages, or other securities are: None.

4. Paragraphs 2 and 3 include, in cases where the stockholder or security holder appears upon the books of the company as trustee or in any other fiduciary relation, the name of the person or corporation for whom such trustee is acting; also the statements in the two paragraphs show the affiant's full knowledge and belief as to the circumstances and conditions under which stockholders and security holders who do not appear upon the books of the company as trustees, hold stock and securities in a capacity other than that of a bona fide owner.

5. The average number of copies of each issue of this publication sold or distributed, through the mails or otherwise, to paid subscribers during the 12 months preceding the date shown above was: 4,433. (Signed) Clark D. Bridges, Business Manager.

Sworn to and subscribed before me this 21st day of October, 1960. (Signed) Lillian Liddy. (My commission expires October 21, 1963.)

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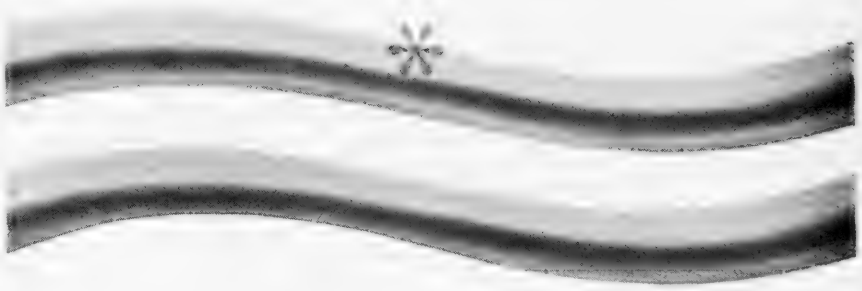

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The Red Carpet at Los Angeles

Hundreds of visitors from other countries who came to attend the Thirteenth International Congress on Occupational Health in New York last July made use of the occasion to visit the United States. Industrial facilities, research centers, and universities were the chief interest points. In at least one instance, a whole community became involved. Los Angeles rolled out the red carpet of hospitality for Richard A. Trevethick, M.D., and B. P. R. Hartley, M.D., industrial physicians with United Steel Company subsidiaries at Sheffield, England.

Under the stimulus of CLARENCE L. LLOYD, M.D., Corporate Medical Director of North American Aviation, Inc., and President of the Western Industrial Medical Association, the visitors were entertained in a whirlwind of wining, dining, sightseeing, and shop-talk. The social highlight of the visit was a banquet attended by more than 300 persons who work in the industrial health field or in closely adjoining areas of professional activity.

The banquet was also used as the occasion for presenting a special plaque, from the Western Industrial Health Conference to DOUGLAS A. CAMPBELL, the distinguished Referee of the California Industrial Accident Commission, Los Angeles. Shown on this page are a few of the photographs made at the banquet.

D. D. McKinnon, M.D., Past President of Western Industrial Medical Association, seated between honored guests, Richard A. Trevethick, M.D., and B. P. R. Hartley, M.D., industrial physicians with United Steel Company subsidiaries in England. Standing, left to right, are Henry G. Morgan, M.D., and Herbert J. Kirchner, M.D., Industrial Section of Los Angeles County Medical Association; Clarence L. Lloyd, M.D., and Edward J. Zaik, M.D., officers of the Western Industrial Medical Association.

The banquet was attended by more than 300 persons.

Left to right: Douglass A. Campbell, J.D., Referee, California Accident Commission, Los Angeles; Rachael Richter, R.N., Todd Company, Division of Burroughs Corp., Los Angeles; Dr. Trevethick; Catherine Colling, R.N., Standard Oil Co., San Francisco; Florence Piazza, R.N., Ampec Corp., San Carlos, Calif.; Dr. Hartley; Mildred Brown, R.N., Director, School of Nursing, Los Angeles County General Hospital; Lois Tate, R.N., Collier Carbon & Chemical Corp., Brea, Calif.; Elnora Ashby, R.N., Douglas Aircraft Co., El Segundo, Calif.

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